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## HEALTH NEEDS OF THE NATION<sup>1</sup>

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There are several reasons why I can bring before you problems concerning the health needs of the Nation in an entirely different spirit from that in which I would address any other group in this country.

In the first place, you of the Red Cross are realistic. You are accustomed to face truth squarely. You cope with disaster on its own terms.

Further, you have more than an academic interest in truth. You are accustomed to action. I have never seen any real need presented to you which did not meet with swift and constructive response.

More than that, though you carry on a great tradition, you are not bound by tradition in your attack upon a problem. You have a flexibility of approach that makes the planting of gardens to prevent the disaster of pellagra as orthodox an example of Red Cross method as service to the war veteran. Because of this you have evolved into an organization which can be geared to the needs of peace no less than war; and can understand the necessity of national fitness in terms of both.

It is for these reasons that I come before you today to consider some of the work which needs to be done by all of us who are concerned with national fitness, as well as to assure you of continued interest and cooperation in the work you now do.

It is true that great progress has been made toward better health in some categories. Since 1900, the death rate from all communicable diseases has dropped by 70 percent, from 383 to 116 per 100,000 population. The general death rate from all causes has gone down 34 percent. If the death rates of 1900 had continued to prevail last year, there would have been 817,745 added deaths in the Nation.

Nothing is easier than to be complacent about this. We have learned a good deal about the great plagues of the generation just past. Except for syphilis, which we have just begun to fight, most

<sup>1</sup> Address delivered at the annual Red Cross Convention, Washington, D. C., April 25, 1939.

of the epidemics are behind us, because we feared them the most and fought them the hardest, because it seemed so dreadful to see men, women, and children stricken suddenly, die swiftly, and together. No community once hard hit by yellow fever, cholera, or smallpox, spared money, men, or effort to save themselves from such a tragedy again. As soon as scientific knowledge was found to fight such diseases, it was put to work. The lag was short between the discovery of such knowledge and its application.

In this year of 1939, however, we find ourselves confronted with an entirely different situation. We have learned a good deal about diseases other than the epidemics, other diseases which kill and maim and lay waste, and load a staggering burden of expense upon the community. But in spite of the fact that there is scientific knowledge about them which is just as effective as our knowledge about yellow fever, we do very much less than we did because of fear of yellow fever or similar plagues. Partly, I think, because it frightens us less when people die one at a time, separately, of many and various maladies, particularly those of which we have no fear for ourselves or our families. Then, too, we no longer see the sick people about us. Though more than a hundred thousand persons die from the pneumonias every year, it does not seem a major disaster; because, in modern life, we seldom take personal responsibility for the sickness or misfortune of our neighbor. Or, if our fellow townsman falls sick of a cancer, and he cannot afford the complicated and expensive professional services which might cure it, we expect him to apply to the proper public authorities. But if the public authorities are not empowered to do anything about cancer, or if they have no budget to do anything, or if there are no facilities—no X-ray, no radium, no pathological laboratories, no highly specialized cancer surgeons—it finally works out that nobody is responsible. Through a succession of such incidents, another 145,000 lives are lost of which at least a third—more than 48,000 every year—could be saved for useful, productive living.

I could give you innumerable examples such as this which would be typical of a score of preventable diseases we endure. For the less gratifying side of the Nation's health picture is that the increase in the life span comes chiefly from the larger number of children who live through the once-dreaded second summer. There is little increase in the number of men and women who live, and are well, through middle life. Undramatic, often preventable diseases continue to take their toll. I have mentioned pneumonia and cancer. We do not yet know how to prevent them; but if, without counting the contents of their pocketbooks, the victims could have prompt and good, modern medical care, the most conservative doctors I know estimate the saving in life from these two causes alone to be large enough to populate a

city the size of Albany. If these people lost their lives from a flood, all at once, all together, if that number of lives were lost every year, the Red Cross and the Nation at large would raise untold millions to prevent it, even though it meant changing the whole course of a river or leveling a range of mountains.

Syphilis is 86 percent curable in its early stages. Yet it is practically unchecked except in a few communities making a determined stand against it. To them this year we were able to give some real help from the Federal Government. Moreover, other than the inattention of governments, State and local, there is no reason for tuberculosis to be five times more prevalent in some communities than it is in others. There is no reason for deaths and sickness from diphtheria to continue to occur when some counties and cities have been able to stamp it out entirely. There is no reason for twice as many babies to die each year in certain backward communities as in those where a modern health program is in force. It is even less logical for the decline of preventable disease to be less and the death rates to be higher among the farmers, where health service is poor, than in crowded industrial cities where good health service is available. Neither does there seem much sense to the fact that the sick citizen who is poor suffers a higher death rate from practically all causes.

The blot on our national fitness because of preventable disease is sad enough. Even less cheering is the evidence of malnutrition prevalent in a land of plenty. Your children and mine, the college-entrance records show, are on the average taller than their parents and physically better specimens; because, among other reasons, science has taught us how to feed them better. I hope their mental equipment also is proportionately better, but of that I have no present proof. But step by step, as the income level goes down among millions of children and youths whom this country needs for the arts of peace and the defenses of war, you will find, because of underfeeding and improper feeding, stunted growth, imperfections in development, subnormal height, weight, and muscular tone, and a *proved* lack of mental alertness. And I speak only of the children, because malnutrition does irremedial damage to them. There also are millions of adults half fed and almost wholly physical failures.

Here then, in scanty outline, is a present pressing problem of our Nation. I need not belabor the point, in speaking to a great humanitarian organization such as this, that the human needs of our people should take precedence over some of the deficiencies in our physical and administrative set-up. They arise from our failure to check preventable disease when we know how to do it, and our failure to feed children abundantly and correctly when we have the food to do it. Surely, the practical thing to do is to correct these failures.

Official action to deal with this situation which has been my concern can go no faster or further than the understanding of the people about it or their voluntary wish to act effectively. It must be a joint effort. And your organization has forgotten more than anyone else ever knew about arousing citizen interest and organizing voluntary action.

Most of you have heard, at least in general, of the National Health Program, which aroused enthusiastic support from consumers' groups especially, when it was discussed last summer at the National Health Conference called by the President's Interdepartmental Committee to Coordinate Health and Welfare Activities. There has been considerable difference of opinion as to the precise methods which would be most effective, but also a considerable unanimity as to objectives among all groups concerned, professional and civic. Deliberately planned as an evolutionary development over a 10-year period, the aims are these:

To strengthen and extend public health services—Federal, State, local—throughout the country as our central lines for defense and advance.

To promote a frontal attack on causes of sickness and death for the control of which public health has effective weapons.

To encourage research and experimentation that will broaden the effective attack on ailments which science has yet to master.

To protect motherhood and conserve childhood, for their well-being is essential to our future as a people.

To expand the physical facilities for good health—hospitals, sanatoria, laboratories, health centers—which are the workshops and scientific aids of modern practice.

To protect patients and their families against the hazards of unemployment and wage loss due to sickness.

To bring adequate medical care within the reach of everyone.

Citizens everywhere are beginning to appreciate the fact that there is not only a human but an economic value in the steps we take forward to improve the national health. I am not an economist, but I think it is a perfectly logical assumption that a healthy consuming nation encourages a healthy production rate and a higher percentage of normal employment. You undoubtedly have noticed that in the pattern of our past experience in America, any permanent upswing from industrial depression resulted from the discovery and fulfillment of new needs for the people. Our balance sheets might tell a very different tale from that which they now do if we actually set to work to transform this population from a status which is half sick and half well, and in which those who are well must carry the whole burden of caring for the sick, the disabled, and those dependent and delinquent because of sickness.

We need to solve the problem of getting the food that we have to those who must have more food and more diversified food. We need to avoid the economic waste of sickness we can cure and death we

can avert from the fruitful years. These are great ambitions. The means of achieving them are accumulating in our hands. The task will not be done today, or tomorrow, but I think the temper of the people makes it inevitable that it will be done. I believe it will be done more wisely, more in accordance with the American tradition, if, with the technique by means of which you, the American Red Cross, have evolved a great instrument for meeting national disasters which have descended swiftly and without warning, you would assume responsibility for forging an instrument to deal with this situation, which we have come to think of as chronic for the Nation, but which is catastrophic for the individual and the family.

It is particularly in the field of better nutrition that I believe a broadly conceived, dynamically executed policy under the aegis of the American Red Cross might transform our whole national status. In saying this I am not unaware of the nutrition work in which you have pioneered. It is because of your success in fighting pellagra in the South, because of your work in the dust bowl, your contributions to nutrition programs of the visiting nurse, the school teacher, and the public health agency, that I think of you as the great potential instrument for utilizing the vast and valuable volunteer effort without which official action alone can never accomplish enough. The job is threefold; as I see it. It means re-education of millions in the use of foods. It means the machinery for getting food into empty hands, and finally, it means a vast multiplication and improvement of all present effort to grow gardens where there are none, to make sure that every child has the needed quart of clean milk a day, and to use every possible device that will encourage the individual to enrich his own diet by the means ready at his hand.

Here and there many people are doing a bit of this. From the all-essential point of view of stimulating and guiding voluntary effort, local and national, there is no other agency which can tie the strands together as well as you. There is no other agency which can give such national visibility to the stark and simple fact that millions hunger in a land of plenty. There is no other group of men and women in America which can contribute so much, if you will, to the clarity and quality of our official thinking, as each of us moves ahead to his designated sector of the appointed task.

There are so many and such great problems confronting us today about which, at the moment, there seems not much that we can do. Here are some great human needs which we know how to satisfy. If we do satisfy them, the impact will be far reaching. I have never heard it intimated that in any respect the results would be other than beneficent. All that we need is the impulsion to carry through a long-range, laborious job, the ability to integrate work now being done so that no effort is wasted, and the desire to lift our sights and

raise our performance to a higher level than we have heretofore known.

Much of what is needed to do the job, the American Red Cross can give to America. I hope that public health may look forward to your leadership.

## STUDIES OF SEWAGE PURIFICATION

### X. CHANGES IN CHARACTERISTICS OF ACTIVATED SLUDGE INDUCED BY VARIATIONS IN APPLIED LOAD\*

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In a number of earlier papers from this laboratory and elsewhere (1, 2, 3), studies of oxidation characteristics of activated sludge have been reported. It was found that the activated sludge alone was oxidized at a rather uniform rate. As soon as sewage was added the rate of oxygen utilization was increased, because of the oxidation of the nutrients in the sewage, by the tremendous bacterial population in the sludge. It was shown (4, 5) that the oxygen requirement of the sludge alone and its capacity for increased oxidation when given food were important characteristics of activated sludge. A later paper (6) presents the results of a study in which the rate of total purification of sewage and its rate of oxidation were observed simultaneously. The proportion of the total purification that was accounted for by oxidation was determined. The difference between the quantities of the total carbonaceous Biochemical Oxygen Demand (L value) of the substrate that is oxidized and that is removed by activated sludge was referred to as the net adsorption and synthesis.

In this paper are presented the results of experiments in which a small activated-sludge plant was operated with varying loads to determine, for purposes of plant control, the value of such criteria of sludge condition as rates of total purification and of oxidation. These data supplement our previous conclusions concerning the importance of the quantity of oxygen required and the capacity of sludge to oxidize substrate, as indices of sludge condition.

#### EXPERIMENTAL METHODS

In the first test the sewage load was increased periodically on a small experimental activated-sludge plant by the procedure of systematically diminishing the aeration time. The plant was first stabilized by operation on a 12-hour fill-and-draw aeration period, and was then changed to a continuous-flow basis with the aeration time periodically reduced at successive intervals to 7.9, 5.8, 4.5, 3.2, and 2.3 hours.

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The length of operation on each aeration interval is indicated in figure 1. After 42 days of operation a break-down in the sewage flow occurred, requiring the termination of the experiment.

During the experimental period, data of plant operation were collected daily. The analyses made included suspended solids and 5-day Biochemical Oxygen Demand (B. O. D.) of the influent sewage; temperature, hydrogen-ion concentration (pH), settleable solids and suspended solids of the aeration tank mixed liquor; percent ash of the sludge and 5-day B. O. D. of the effluent. From these analyses, the sludge index; the plant load, and the percentage reduction of the 5-day B. O. D. by the treatment plant were calculated. The plant load is expressed as pounds of 5-day B. O. D. per day introduced by the sewage per pound of suspended solids in the aeration tank<sup>1</sup> mixed liquor.

In these experiments the plant load was computed by the formula

$$\text{Plant load} = \frac{\text{5-day B. O. D. (p. p. m.)} \times \text{sewage flow (liters) in 24 hours}}{\text{Mixed liquor suspended solids (p. p. m.)} \times \text{capacity of aeration tank (liters)}}$$

For example, for this experimental plant with an aeration tank capacity of 205 liters, on December 4, 1936, the sewage flow was 1,217 liters in 24 hours with a 5-day B. O. D. of 212 p. p. m. and the aeration tank mixed liquor suspended solids averaged 2,568 p. p. m. Therefore, the plant load was

$$\frac{212 \times 1,217}{2,568 \times 205} = 0.490.$$

All of the above operation data are presented in table 1 and are plotted in figures 1 and 2. Figure 1 shows that there was very little change in the pH of the sewage or of the aeration mixture during this period. The temperature of the aeration mixture slowly decreased during the experiment from between 10° and 12° C. at the beginning to between 6° and 8° C. at the end. The suspended solids in the aeration mixture were maintained at between 2,100 and about 2,700 p. p. m. throughout the experimental run.

The percentage by weight of ash in the sludge decreased from around 30 percent at the beginning of the experiment until a minimum value of about 18.5 percent was reached on the twenty-sixth day after the plant had been operating on a 3.2-hour period for several days. From this point on the ash content fluctuated considerably, and the minimum value of 16.3 percent for the entire experiment was obtained on the forty-first day.

<sup>1</sup> It was realized that the solids in the entire system should be included in load calculations. However, measurements indicated that the quantities of suspended solids in the final settling tank were a minor factor (less than 5 percent); consequently, for simplicity, aeration tank solids only were used in these load computations.

At the beginning of the experiment the settling quality of the sludge was poor, but it improved slowly until the sludge index reached 19.3 at the same time that the minimum value of 18.5 percent ash was obtained. These results are contrary to the general rule that the sludge index (calculated as the ratio of suspended solids in p. p. m. to the volume in ml. to which one liter of sludge settles in 30 minutes) is directly proportional to the percentage of ash in the sludge. As in

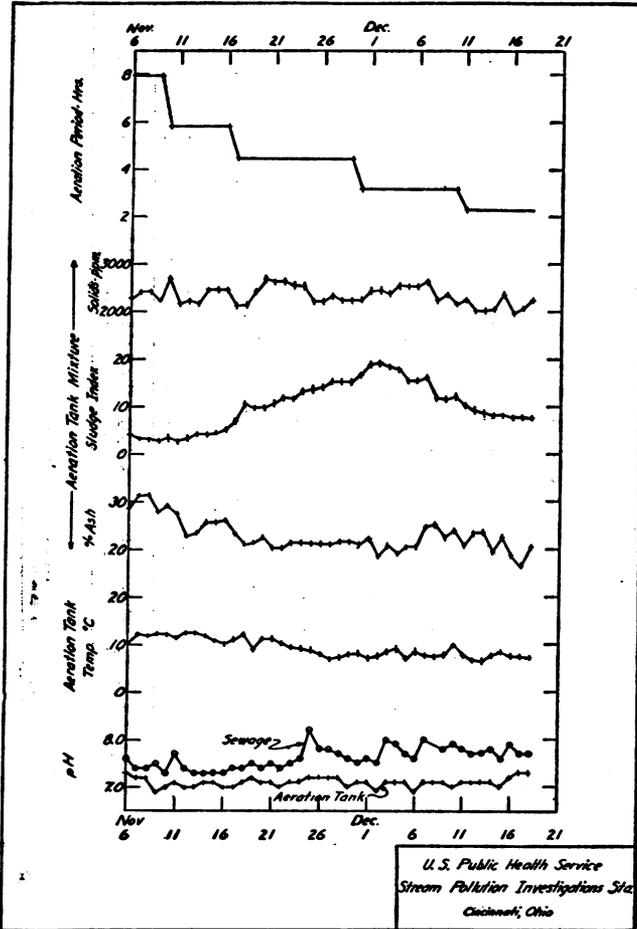


FIGURE 1.—Plant operation data. Aeration period and aeration tank mixed liquor characteristics.

this experiment an inverse relationship existed during the first 26 days, special conditions must have upset the general relationship between the percentage of ash in the sludge and the sludge index. This unusual phenomenon has been noted before at this station.

Figure 2 shows that the suspended solids content of the sewage influent varied from about 85 to 165 p. p. m. during the experiment;

TABLE 1.—Plant operation data, experiment 1

Date (1936)	Influent sewage			Activated sludge aeration mixture						Plant effluent 5-day B. O. D.	Plant loading, lbs. 5- day B. O. D. per day per lb. suspended solids in aeration mixture	Per- cent reduction in 5-day B. O. D.
	pH	5-day B. O. D., p. p. m.	Sus- pended solids, p. p. m.	Tem- pera- ture (°C.)	pH	Settle- able sol- ids in 30 min. (cc./liter)	Sus- pended solids, p. p. m.	Per- cent ash in sludge	Sludge index			
Nov. 6	7.6	133	88	10.3	7.3	560	2,284	28.7	4.1	7.2	0.140	94.6
7	7.4	83	82	12.3	7.2	705	2,408	31.5	3.4	4.0	.083	95.2
8	7.4	83	82	12.0	7.2	765	2,408	31.5	3.2	4.0	.083	95.2
9	7.5	190	160	12.3	6.9	758	2,216	28.0	2.9	7.5	.206	56.0
10	7.3	114	94	12.2	7.0	786	2,696	29.4	3.4	5.8	.139	84.9
11	7.7	225	121	11.5	7.1	758	2,166	27.6	2.9	7.9	.340	66.5
12	7.4	152	120	12.5	7.0	670	2,232	22.9	3.3	10.3	.223	93.2
13	7.3	165	106	12.5	7.0	503	2,184	23.6	4.3	17.0	.247	87.9
14	7.3	141	100	12.0	7.1	608	2,472	25.9	4.1	3.1	.187	97.8
15	7.3	141	100	11.0	7.1	573	2,472	25.9	4.3	3.1	.187	97.8
16	7.3	149	120	10.3	7.0	477	2,464	26.1	5.2	10.2	.198	93.1
17	7.4	155	119	11.0	7.0	303	2,112	23.5	7.0	4.8	.318	96.9
18	7.4	183	108	12.2	7.1	200	2,136	21.0	10.6	6.7	.282	96.3
19	7.5	256	108	9.0	7.2	245	2,412	21.4	9.8	17.1	.248	102.2
20	7.4	174	118	11.3	7.1	273	2,688	22.6	9.9	4.4	.276	91.3
21	7.5	268	104	11.3	7.1	247	2,640	20.3	10.7	6.9	.440	97.4
22	7.4	268	104	10.3	7.0	220	2,640	20.3	12.0	6.9	.440	97.4
23	7.5	---	167	9.5	7.1	217	2,552	21.7	11.8	---	---	---
24	7.6	134	140	9.2	7.1	193	2,544	21.5	13.2	14.3	.228	89.4
25	8.2	185	134	8.8	7.2	162	2,216	21.9	13.7	2.7	.362	98.5
26	7.8	185	134	8.0	7.2	158	2,216	21.3	14.0	2.7	.362	98.5
27	7.8	160	124	7.0	7.2	155	2,344	21.2	15.1	8.4	.296	94.7
28	7.7	145	122	7.3	7.2	147	2,240	21.8	15.2	4.5	.280	96.9
29	7.6	145	122	8.0	7.0	143	2,240	21.8	15.7	4.5	.280	96.9
30	7.5	179	138	8.0	7.1	133	2,240	21.1	16.8	14.3	.473	92.0
Dec. 1	7.6	190	120	7.2	7.1	128	2,432	22.4	19.0	10.5	.463	94.5
2	7.5	77	100	7.5	6.9	127	2,452	18.5	19.3	7.7	.186	90.0
3	8.0	169	100	8.7	7.1	130	2,388	20.9	18.4	7.5	.419	95.6
4	7.9	212	136	9.0	7.1	143	2,568	19.9	18.0	8.7	.490	95.9
5	7.7	119	92	7.0	7.1	163	2,536	20.7	15.5	6.3	.278	94.7
6	7.6	119	92	8.5	6.9	163	2,536	20.7	15.6	6.3	.278	94.7
7	8.0	237	106	7.7	7.1	163	2,632	24.9	16.1	10.1	.535	95.7
8	7.9	174	72	7.5	7.1	187	2,236	25.2	11.9	10.5	.463	94.0
9	7.8	139	73	7.8	7.1	203	2,364	22.7	11.7	9.4	---	93.2
10	7.9	192	94	10.0	7.0	180	2,176	24.1	12.1	11.0	.523	94.3
11	7.8	185	96	7.7	7.1	220	2,268	20.9	10.3	12.4	.694	93.3
12	7.7	340	84	6.7	7.1	220	2,012	23.7	9.2	6.1	1.420	58.4
13	7.7	340	84	6.5	7.1	223	2,012	23.7	8.8	6.1	1.420	58.4
14	7.8	294	118	7.7	7.1	247	2,056	19.5	8.3	15.9	.598	93.2
15	7.6	133	137	8.3	7.0	287	2,500	22.7	8.2	13.8	.475	89.6
16	7.9	218	113	7.5	7.2	253	1,960	18.6	7.8	10.4	.934	95.2
17	7.7	259	115	7.5	7.3	263	2,084	16.3	7.9	16.4	1.040	92.7
18	7.7	152	108	7.2	7.3	285	2,232	20.6	7.8	15.3	.572	90.0

<sup>1</sup> Dissolved oxygen depleted after incubation in these samples; consequently the B. O. D. values are low.

<sup>2</sup> These results are too high because effluent B. O. D. values are low.

also that the 5-day B. O. D. of this sewage fluctuated considerably from a minimum value of about 77 p. p. m. to a maximum of 340 p. p. m. The plant loading, calculated in pounds of 5-day B. O. D. per pound of suspended solids in the aeration mixture, has been plotted in figure 2, which clearly shows the trend of increasing load during this experiment. The percentage of 5-day B. O. D. reduction obtained on the sewage passing through the plant varied between 88 and 98.5, but there was no regular decrease corresponding with increases in plant loading.

An examination of these operating data does not reveal the point at which overloading began. On the basis of sludge index it may appear

that for a time the increased loading benefited the sludge. None of these common indices, however, gives any indication as to when the sludge reached an optimum biochemical condition from the standpoint of sewage purification or shows when the sludge was definitely overloaded.

An evaluation of the changes taking place in the biochemical quality of the sludge during this loading experiment was attempted

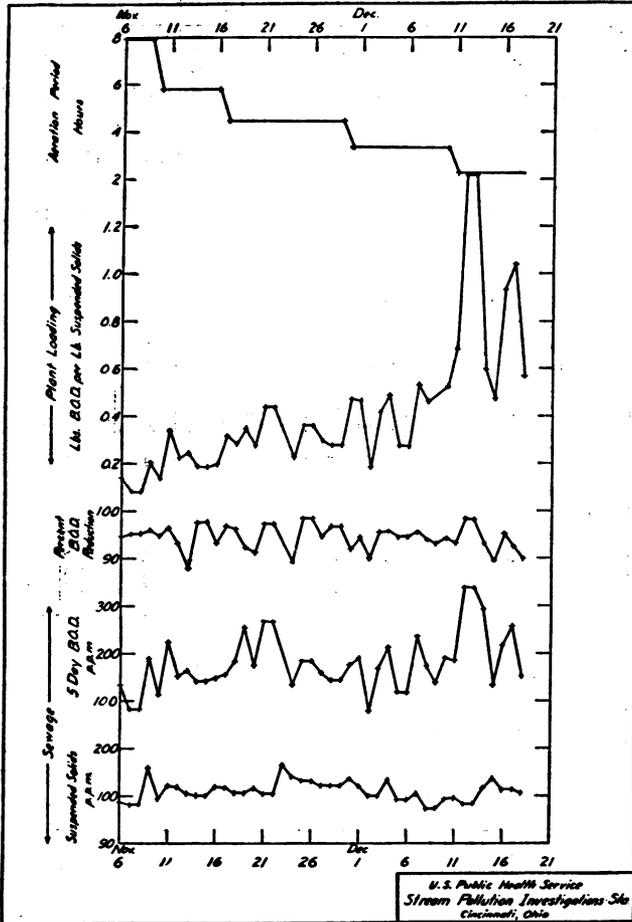


FIGURE 2.—Plant operation data. Sewage strength, purification, and plant loading.

by means of the sludge and substrate-feed mixture oxidation tests and 20-day substrate B. O. D. removal test. Eight-liter samples of sludge were taken from the plant periodically for this purpose and the tests were made as described (5, 6) previously. Twelve series of these tests were run upon sludge removed from the plant during this experiment.

The substrate B. O. D. removal tests were made by adding the sewage feed to the settled solids from a 6-liter sample of the aeration mixture from the tank. The sewage and the sludge were brought to 20° C. and, after mixing, were aerated at this temperature in the laboratory for 5 hours. The 5-day and 20-day B. O. D. were determined on the old supernatant removed when the aeration mixture was taken and on the sewage feed added. These values for all tests are given in table 2. The initial B. O. D. of the mixed substrate was calculated from the B. O. D. of the old supernatant and that of the sewage added, as shown in table 2. After 1½, 3, and 5 hours, supernatant was removed from the aeration mixture in the test bottle and the 5-day and 20-day B. O. D. determined. From the values of B. O. D. remaining in the supernatant after these aeration periods

TABLE 2.—Results of substrate B. O. D. removal tests of plant sludge on domestic sewage, experiment 1

Days after start of experiment.....	0	3	7	10	14	17	20	23	27	31	34	38
Test number.....	1	2	3	4	5	6	7	8	9	10	11	12

DILUTION METHOD, 5-DAY B. O. D. OF SUBSTRATES (P. P. M.)

Old supernatant.....	6.2	15.5	17.6	13.3	29.3	18.7	16.2	21.2	12.6	16.6	32.7	24.2
Sewage feed.....	202.0	189.0	249.0	234.0	224.0	213.0	229.0	229.0	256.0	222.0	316.0	104.0
Initial supernatant substrate <sup>1</sup> .....	123.7	119.6	156.4	145.7	146.1	135.3	143.9	140.5	158.6	139.8	202.7	2.1

Supernatant substrate after—

1½ hours aeration.....	25.3	24.9	17.8	19.6	21.6	33.7	22.3	31.7	34.7	35.3	48.7	28.7
3 hours aeration.....	17.2	16.0	13.5	13.4	15.8	19.4	14.0	19.2	25.4	37.8	21.7	22.7
5 hours aeration.....	12.4	10.7	11.3	9.9	17.3	19.0	15.4	21.2	24.1	25.7	27.5	19.2

DILUTION METHOD, 20-DAY B. O. D. OF SUBSTRATES (P. P. M.)

Old supernatant.....	13.0	24.1	47.9	26.9	44.6	43.4	56.1	61.1	68.8	67.3	83.2	85.5
Sewage feed.....	365.0	524.0	455.0	388.0	455.0	426.0	549.0	457.0	666.0	656.0	756.0	353.0
Initial supernatant substrate <sup>1</sup> .....	224.2	324.0	292.2	243.6	290.8	273.0	351.8	298.6	427.1	420.5	486.9	246.0

Supernatant substrate after—

1½ hours aeration.....	123.6	114.1	116.8	74.6	101.5	141.9	138.4	159.9	227.2	231.2	292.0	149.1
3 hours aeration.....	112.2	88.7	75.2	74.9	68.7	75.3	116.0	128.7	185.0	177.0	193.0	150.2
5 hours aeration.....	73.5	81.2	41.5	19.5	30.6	62.0	62.2	92.8	82.6	167.7	166.2	157.2

PERCENTAGE REDUCTION OF 20-DAY B. O. D. SUBSTRATE

After 1½ hours aeration....	43.5	64.8	60.0	69.4	65.1	48.0	60.7	46.5	46.8	45.0	40.0	39.4
After 3 hours aeration....	50.0	72.6	74.3	69.3	76.4	72.4	67.0	56.9	56.7	57.9	61.0	38.9
After 5 hours aeration....	67.2	74.9	85.8	92.0	89.5	77.3	82.3	68.9	80.7	60.1	65.9	36.1

<sup>1</sup> Each value represents the quantity of oxygen required by 0.6 liter of sewage plus 0.4 liter of old supernatant.

and the initial B. O. D. of the substrate, the percentage of the 20-day substrate B. O. D. that was removed was calculated and is also given in table 2.

The substrate B. O. D. removal performance of the sludge obtained during these tests is shown in a series of curves in figure 3. During the first 4 tests, representing the first 10-day operation period, the 5-day substrate B. O. D. removal test showed no change in the sludge. The next 4 tests showed a slightly lowered efficiency in the 5-day substrate removal performance. In the last 4 tests the 5-day substrate B. O. D. removal capacity of the sludge became further impaired. However, at no time during these tests was a striking change indicated in the 5-day B. O. D. removal performance of the sludge in spite of

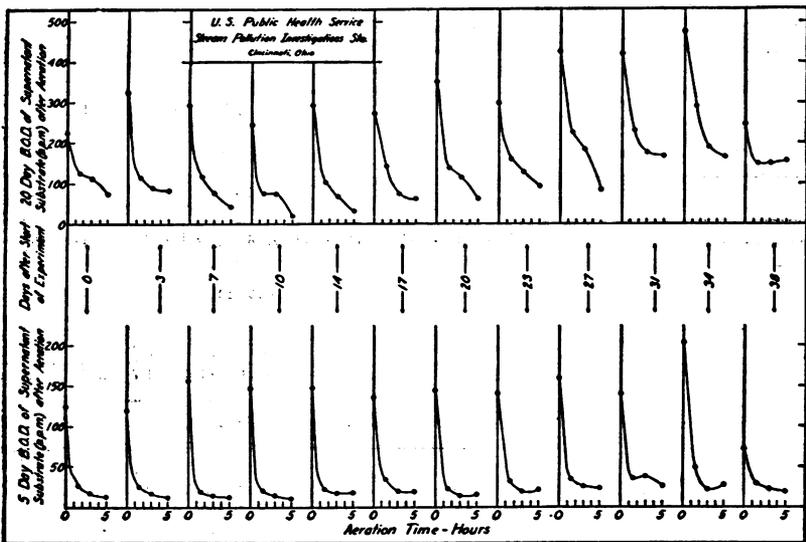


FIGURE 3.—Efficiency of sludge as measured by decrease in B. O. D. of supernatant substrate during aeration in a closed system.

increasing loads put on the plant. This probably may be interpreted as indicating that the 5-day B. O. D. removal test on sludge is a rather poor and insensitive indicator of the biochemical condition of the sludge.

An examination of the results obtained in the 20-day substrate B. O. D. removal tests, which are also plotted in figure 3, indicates that this test is a better indicator of change in sludge condition than the 5-day B. O. D. removal test. However, because of the long incubation time required, it has no practical value in plant operation.

The data for the oxidation tests performed during this experiment are given in table 3. The data for the oxygen required for the sludge control and for the sludge-plus-feed mixture are plotted in figure 4. These curves show that the oxygen requirement and the rate of oxygen utilization of the control sludge during the first 3-hour aeration period increased tremendously as this experiment progressed. While there was some increase in the rate of oxygen utilization of the sludge-feed mixture in the tests until the twenty-seventh day of the experiment, this did not keep up with the increasing rate in the control sludge. Consequently, the difference in the quantities of oxygen utilized by these systems, which difference represents substantially the oxidation of the substrate, declined after the tenth day of the experiment. This will be observed in figure 5, where the oxygen results obtained, as described for substrate oxidation, are plotted. An examination of figures 4 and 5 indicates that the biochemical

TABLE 3.—Results of oxidation tests with plant sludge on domestic sewage, experiment 1

Days after start of experiment.....	0	3	7	10	14	17	20	23	27	31	34	38
Test number.....	1	2	3	4	5	6	7	8	9	10	11	12
Suspended solids in sludge, p. p. m.....	2, 372	2, 460	2, 252	2, 576	2, 596	2, 336	2, 360	2, 140	2, 392	2, 444	2, 080	3, 364
<b>MG. OXYGEN USED BY CONTROL MIXTURE (B)</b>												
1½ hours aeration.....	15.0	18.6	27.4	21.0	35.0	23.2	29.2	27.5	51.6	37.8	38.5	55.0
3 hours aeration.....	27.1	32.4	50.2	42.2	62.1	40.1	57.5	59.8	101.2	75.5	71.5	117.8
5 hours aeration.....	36.6	48.4	78.9	50.8	100.7	68.7	98.4	98.8	150.2	105.9	109.9	165.0
<b>MG. OXYGEN USED BY FEED AERATION MIXTURE (A)</b>												
1½ hours aeration.....	-----	51.1	56.6	62.8	57.4	47.5	57.1	59.5	70.5	47.8	69.5	64.9
3 hours aeration.....	75.5	83.2	98.7	98.0	94.8	87.0	100.7	103.0	133.7	92.3	115.8	118.6
5 hours aeration.....	89.1	116.9	136.2	143.4	141.4	131.0	165.9	156.6	203.9	133.8	158.3	156.5
<b>C. MG. OXYGEN USED TO OXIDIZE SUBSTRATE FEED (A-B)</b>												
1½ hours aeration.....	-----	32.5	29.2	41.8	22.4	24.3	27.9	32.0	18.9	10.0	31.0	9.9
3 hours aeration.....	48.4	50.8	48.5	55.8	32.7	46.9	43.2	43.2	32.5	16.8	44.3	0.8
5 hours aeration.....	52.5	68.5	57.3	92.6	40.7	62.3	67.5	57.8	53.7	27.9	48.4	-8.5
<b>D. PERCENTAGE OF 20-DAY SUBSTRATE FEED B. O. D. OXIDIZED<sup>1</sup></b>												
1½ hours aeration.....	-----	10.3	10.7	18.0	8.2	9.5	8.5	11.7	4.7	2.5	6.8	4.7
3 hours aeration.....	22.1	16.2	17.8	24.0	12.0	18.3	13.1	15.8	8.1	4.3	9.8	0.4
5 hours aeration.....	24.0	21.8	21.0	39.8	14.9	24.4	20.5	21.1	13.4	7.1	10.7	-4.0
<b>E. MG. OXYGEN PER GRAM SUSPENDED SOLIDS IN CONTROL MIXTURE</b>												
1½ hours aeration.....	6.3	7.6	12.2	8.2	13.5	9.9	12.4	12.9	21.6	15.5	18.5	16.3
3 hours aeration.....	11.4	13.2	22.3	16.4	23.9	17.2	24.4	27.9	42.3	30.9	34.4	35.0
5 hours aeration.....	15.4	19.7	35.0	19.7	38.8	29.4	41.7	46.2	62.8	43.3	52.8	49.0

<sup>1</sup> This is obtained by dividing the C values by 0.6 of the 20-day sewage feed values in p. p. m. as given in table 2 and multiplying by 100. (There were 600 ml. of sewage used as feed.)

qualities of the sludge were not injured and may have been benefited somewhat by increased load until the tenth day of the experiment. A decided decrease in the substrate oxidation capacity is indicated

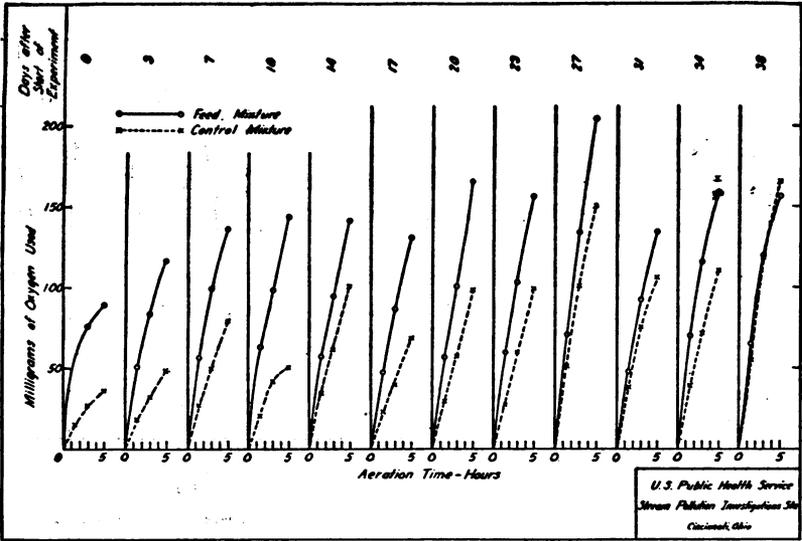


FIGURE 4.—Oxygen utilization of control sludge and sludge with added sewage measured while undergoing aeration in a closed system.

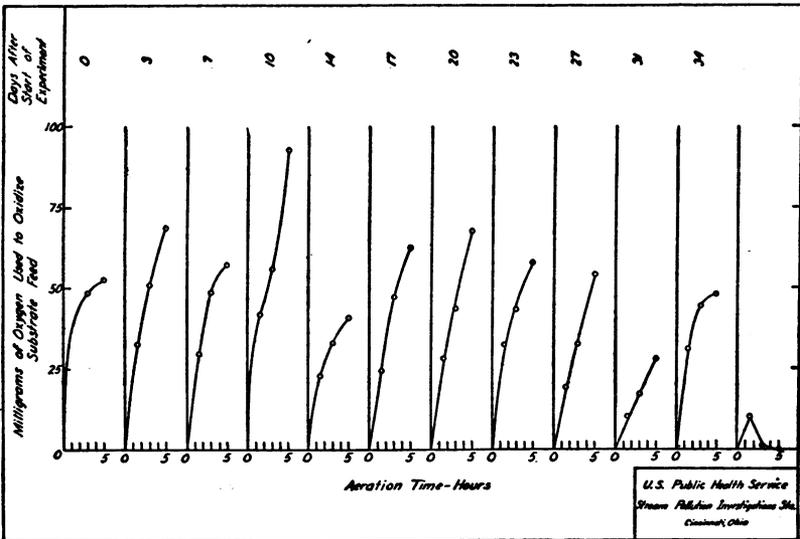


FIGURE 5.—Oxidation of substrate feed by activated sludge as measured by the differences in oxygen utilization of control sludge and sludge with added sewage in closed aeration systems.

by the fifth test on the fourteenth day and, although some recovery is indicated in the following 2 tests, there was, with one exception, a consistent decrease in the oxidation capacity of the sludge thereafter.

On the final test, the sludge control required as much oxygen during the 3- and 5-hour aeration period as the sludge feed mixture, a condition which indicated a badly overloaded sludge.

Figure 6 shows the oxygen requirement per gram of control sludge

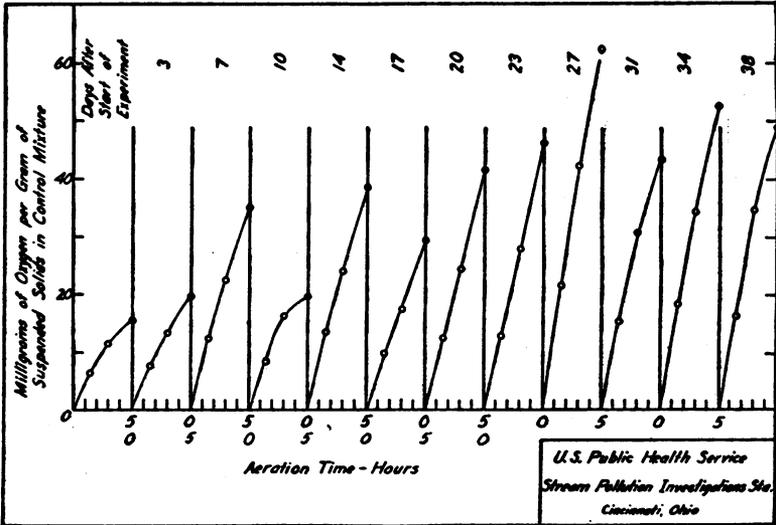


FIGURE 6.—Oxygen requirement per gram of suspended solids in the control sludge.

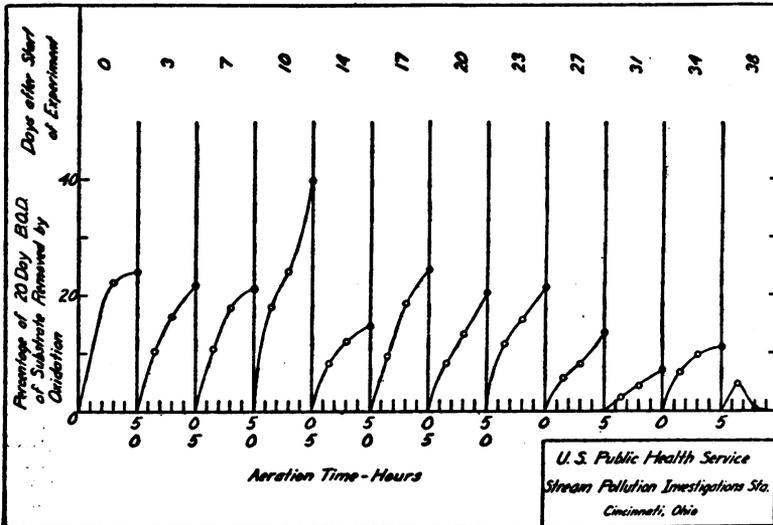


FIGURE 7.—Percentage of 20-day B. O. D. of sewage feed oxidized by activated sludge.

during this experiment. This parameter shows that there was a general increase in the oxygen requirement per gram of control sludge during the 1½- and 3-hour aeration periods after the tenth day of the

experiment. On the basis of these data it may be concluded that if the control-sludge oxygen requirement per gram exceeds about 12 mg. in 1½ hours or 25 mg. in 3 hours, the danger point for the continued successful operation of the system is being approached. This indicator of activated sludge condition is easily obtained and seems to be an excellent one for practical application in plant control. The curves in figure 7 show the percentage of the 20-day B. O. D. of the substrate that is oxidized by the activated sludge in this experiment. They indicate very strikingly the decreasing efficiency of the sludge for oxidizing substrate during the course of this test.

#### EXPERIMENT ON PROLONGED REAERATION OF SLUDGE

The purpose of the second experiment was to study changes in the oxidizing powers of activated sludge during a prolonged aeration period without feeding. In this experiment the experimental activated sludge plant was operated on an 8-hour aeration period for 8 days to obtain a good, normal activated sludge. During this preliminary period, 4 total purification and oxidation tests similar to those described for experiment 1 were run to determine the characteristics of the sludge. Then the sewage feed was cut off, but the tank aeration mixture was aerated at the normal rate for a period of 13 days. Periodically, during the aeration period, samples of sludge were removed and the tests already described were performed, using domestic sewage as the feed. The results of these tests during this experiment have been summarized in tables 4 and 5, and some of the most significant data indicating change in sludge condition have been plotted in figures 8, 9, and 10. The quantities of oxygen used during the 5-hour aeration period of these tests decreased regularly on both the control sludge (B) and the aeration mixture (A). However, the difference between (A) and (B) gradually became less, indicating again that the ability of the sludge to oxidize the added sewage substrate used in the test was being lost. This is best shown in figure 8, where the quantities of oxygen representing the oxidation of substrate (A—B) have been plotted for each test after the feed was stopped in the aeration tank. This indicates that starving a sludge, while decreasing the demand of the sludge itself, also decreases its ability to oxidize nutrient substrate feed which may be added to it. Once this oxidizing ability is lost by prolonged aeration it can be redeveloped only gradually as is done initially. In this case there was not a great deal of damage done during the first 3 days of aeration following elimination of the feed, but between the third and sixth days there was a very great loss in the ability of the sludge to oxidize sewage. By the ninth day this oxidizing ability was very poor and it had practically disappeared by the thirteenth day.

TABLE 4.—Base data and results of *B. O. D.* removal tests of plant sludge on domestic sewage, experiment 2 (activated sludge aeration without feeding)

Date: October 1936.....	5	6	7	8	9	12	15	19
Aeration period, hours.....	8	Inf. <sup>1</sup>	Inf.	Inf.	Inf.	Inf.	Inf.	Inf.
Days after last feeding.....		0	1	2	3	6	9	13
Suspended solids, p. p. m.....	2,808	2,672	2,464	2,168	2,208	2,204	2,164	1,868
Sludge index.....	29.8	29.8	29.0	27.1	27.6	24.4	24.0	19.6
Percent ash in sludge.....	24.1	23.1	24.9	23.9	25.0	27.6	31.4	30.0

DILUTION METHOD, 20-DAY *B. O. D.* (P. P. M.)

Old supernatant.....	40.0	26.6	33.6	67.0	39.2	51.6	42.7	31.0
Sewage feed.....	491.0	590.0	513.0	613.0	377.0	368.0	329.0	349.0
Initial supernatant substrate <sup>1</sup> .....	310.6	364.6	321.2	394.6	241.4	241.4	214.5	221.8

## Supernatant substrate after—

1½ hours aeration.....	131.0	172.0	146.0	210.0	133.0	124.5	106.0	153.0
3 hours aeration.....	78.7	95.7	99.5	136.0	114.0	86.7	94.6	146.0
5 hours aeration.....		45.9	43.4	28.6	80.6	71.9	71.5	136.0

PERCENTAGE REDUCTION OF 20-DAY *B. O. D.* OF SUBSTRATE

After 1½ hours aeration.....	57.8	52.8	54.5	46.8	44.9	48.4	50.6	31.0
After 3 hours aeration.....	74.7	73.8	69.0	65.5	52.8	64.1	55.9	34.2
After 5 hours aeration.....		87.4	86.5	92.8	66.6	70.2	66.7	38.7

<sup>1</sup> Each value represents the quantity of oxygen required by 0.6 liter sewage plus 0.4 liter old supernatant  
<sup>1</sup> Infinite or continuous aeration of sludge without the addition of feed.

TABLE 5.—Results of oxidation tests with plant sludge on domestic sewage, experiment 2 (activated sludge aeration without feeding)

Days after last feeding.....		0	1	2	3	6	9	13
Test number.....	4	5	6	7	8	9	10	11

## MG. OXYGEN USED BY CONTROL MIXTURE (B)

1½ hours aeration.....	35.1	27.3	12.9	17.3	19.7	18.3		10.3
3 hours aeration.....	53.2	48.8	36.5	32.1	32.1	32.5	27.7	15.9
5 hours aeration.....		71.1	59.4	50.6	45.0	45.8	43.1	22.7

## MG. OXYGEN USED BY FEED AERATION MIXTURE (A)

1½ hours aeration.....	83.4	77.8	59.2	58.9	55.4	23.5	16.3	14.7
3 hours aeration.....	145.9	134.3	92.9	95.0	79.3	42.9	38.3	21.2
5 hours aeration.....		175.1	126.5	120.4	107.5	69.6	61.3	23.6

## C. MG. OXYGEN USED TO OXIDIZE SUBSTRATE FEED (A-B)

1½ hours aeration.....	48.3	50.5	46.3	41.6	35.7	5.2		4.4
3 hours aeration.....	92.7	85.5	56.4	62.9	47.2	10.4	10.6	5.3
5 hours aeration.....		104.0	67.1	66.8	62.5	23.8	18.2	.9

D. PERCENTAGE OF 20-DAY SUBSTRATE FEED *B. O. D.* OXIDIZED<sup>1</sup>

1½ hours aeration.....	16.4	14.3	15.0	11.3	15.8	2.4		2.1
3 hours aeration.....	31.5	24.2	18.3	17.1	20.9	4.7	5.4	2.5
5 hours aeration.....		29.4	21.8	19.0	27.6	10.8	9.2	.4

## E. MG. OXYGEN PER GRAM SUSPENDED SOLIDS IN CONTROL MIXTURE

1½ hours aeration.....	12.5	10.2	5.24	7.98	8.92	8.30		5.51
3 hours aeration.....	18.9	18.3	14.8	14.8	14.5	14.7	12.8	8.51
5 hours aeration.....		26.6	24.1	23.3	20.4	20.8	19.9	12.2

<sup>1</sup> This is obtained by dividing the C values by 0.6 of the 20-day sewage feed values in p. p. m. as given in table 4 and multiplied by 100. (There were 600 ml. of sewage used as feed.)

The quantities of oxygen used per gram by the control sludge in each test are plotted in figure 9. This series of curves shows that the rates and quantities of oxygen used by the sludge gradually decreased

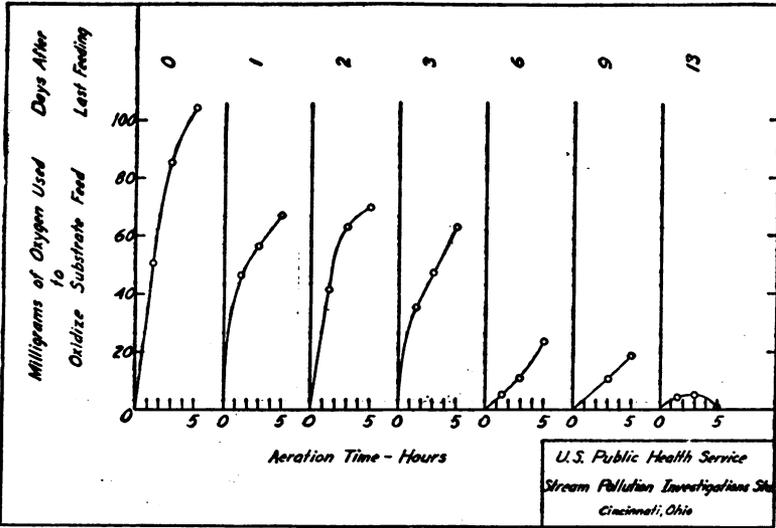


FIGURE 8.—Oxidation of substrate feed by activated sludge in closed system as sludge deteriorated.

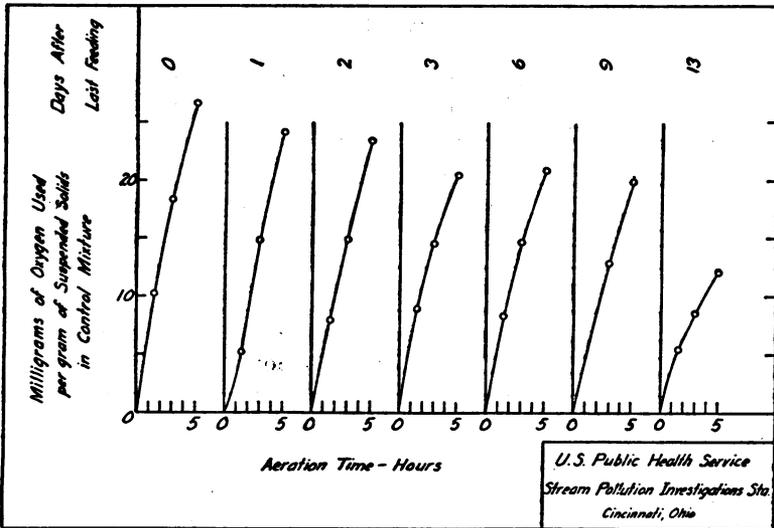


FIGURE 9.—Oxygen requirement per gram of solids in the control sludge during long continued re-aeration.

in this experiment. This is, as would be expected, exactly opposite to the result obtained in the overloading experiment.

The series of curves in figure 10 are similar to those in figure 7 for

the first experiment and show the reduction, which was the result of starvation, in the percentages of the 20-day B. O. D. of the substrate removed by oxidation.

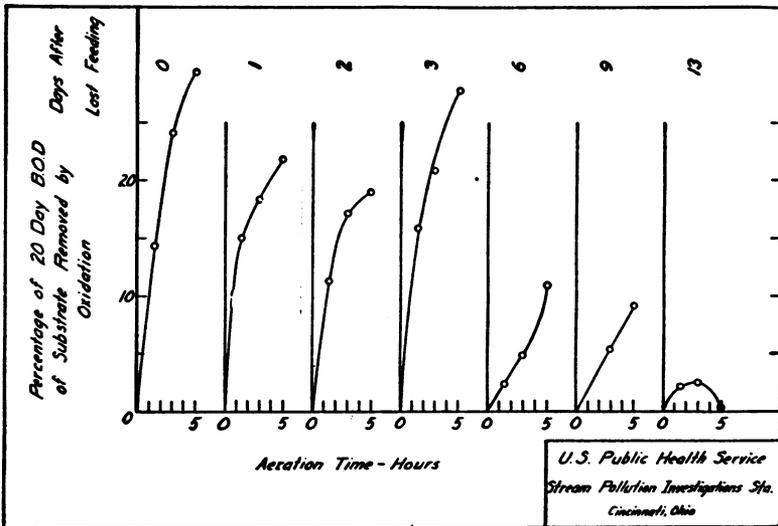


FIGURE 10.—Percentage of 20-day B. O. D. of sewage feed oxidized by activated sludge following various periods of long re-aeration.

#### SUMMARY

In an experiment on overloading of activated sludge and on another with starvation (prolonged aeration without food), suspended solids, sludge index, sludge ash, total B. O. D. removal tests, short-time oxygen demands of sludge, and substrate oxidation tests were used as parameters of change in sludge condition. The experiments indicated that, while the sludge index and sludge ash were important criteria from the settling standpoint, they were very poor indices of change in biochemical quality of the sludge. The B. O. D. removal tests were also poor and insensitive indicators of change in biochemical qualities. The 20-day B. O. D. removal test was superior to the 5-day test as an indicator of past change, but was of no practical value in plant control because of the long incubation period involved.

The short-time oxygen utilization tests on activated sludge appeared to be better indices of change than any of the above parameters. It was shown that the quantity of oxygen used per gram of suspended solids during a short aeration period of the mixed liquor sample undergoing test gradually increased with overloading of the sludge. With cessation of sewage feed to the mixed liquor maintained under continuous aeration, this oxygen use was slowly reduced. Practical tests of this nature can be made easily and this procedure may prove to be

a valuable aid in activated sludge plant control, particularly as an indicator of overloading.

These experiments indicate that the substrate oxidation tests are sensitive indicators of change of biochemical quality of activated sludge. It appears that this most important property of an activated sludge is gradually lost either by prolonged overloading or by starvation. Prolonged aeration without food (sludge re-aeration) altered the normal characteristics of activated sludge to the point where the aerated sludge had entirely lost its ability to oxidize sewage substrate at high rates.

#### ACKNOWLEDGMENT

Acknowledgment is made to former Assistant Chemist P. D. McNamee for much of the analytical work on which this paper is based.

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## A STUDY OF HUMAN SERA ANTIBODIES CAPABLE OF NEUTRALIZING THE VIRUS OF LYMPHOCYTTIC CHORIOMENINGITIS

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The virus of lymphocytic choriomeningitis was first described by Armstrong and Lillie (1) in 1934. Since that date the virus has been recovered in several locations in the United States (2, 3, 4), in England (5), France (6), and Japan (7). In addition to these published reports, two unreported strains have been isolated at the National

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Institute of Health. One of the strains was recovered (by C. A.) from a patient at Gallinger Hospital, Washington, D. C., suffering with clinical lymphocytic choriomeningitis, and the other (by J. G. W.) from guinea pigs that had been inoculated in series with blood of a laboratory attendant suffering with an illness resembling influenza.

Antibodies that are capable of neutralizing the virus of lymphocytic choriomeningitis were demonstrated in human blood sera by Armstrong and Wooley (2) through the medium of the serum-virus protection tests in white mice. Shortly after this, Rivers and Scott (4) and Armstrong and Dickens (8) demonstrated that human blood serum drawn early in the illness failed to neutralize the virus while serum collected late in convalescence did so.

Wooley, Armstrong, and Onstott (9) studied 1,248 human blood sera for protective antibodies against lymphocytic-choriomeningitis virus and demonstrated antibodies in 138, or 11 percent, of them. Fifty-three of the 1,248 sera were from persons on whom a clinical diagnosis of "aseptic meningitis" had been made; and of these, 17, or 32.1 percent, neutralized the virus. The remaining 1,195 sera were from persons who gave no history of central nervous system disease resembling the clinical entity commonly referred to as "aseptic meningitis." Of these 1,195 sera, 121, or 10.1 percent, contained antibodies against the virus of lymphocytic choriomeningitis. These results suggest that contact with the virus of this ailment is widespread in the United States.

The present report deals with a continuation of the study of human sera for the presence of antibodies capable of neutralizing the virus. The choriomeningitis virus employed throughout these tests is the same strain employed in earlier studies (Armstrong (1)), and the technique of the tests is the same as that previously described (9), except that an additional group of 4 mice inoculated with an approximately 1:50 suspension of virus was included, thereby utilizing 16 mice instead of 12 as used in the previous report. The mice largely employed were inbred Swiss stock raised at the National Institute of Health. Occasionally, when the supply of Swiss mice was low, white mice purchased on the open market were used. Only mice weighing between 17 and 20 grams were utilized in the tests.

The study here reported was made on 680 blood sera collected from individuals in various localities in the United States, and from Hawaii, Panama Canal Zone, and Puerto Rico. Among the total of 680 sera, 515 were received from various sources and localities while the remaining 165 were collected by two of us (F. D. S. and J. F. K.).

In table 1 the protection test results for all sera are tabulated by the geographical areas from which they were received. It is noted that 18.1 percent of the total number definitely showed protective antibodies and that the areas represented showed widely divergent

proportions of sera which neutralized the virus. Thus, in the larger groups the proportion showing protection varied from 6.3 percent in the Middle Atlantic States to 44.3 percent in the Pacific States. However, all areas from which a reasonably large number of bloods were collected showed some sera with a degree of protection. These results, when considered with the finding of protection in 11 percent of the 1,248 sera previously reported (9), confirm the belief that antibodies against the virus are widely distributed and that the virus probably infects man more commonly than is generally believed.

TABLE 1.—Results of the protection tests on human sera from various groups of States

	Total number sera	Number of sera showing—				Percent of sera positive
		Strong protection	Moderate protection	Questionable protection	No protection	
New England States.....	6	1	1	-----	4	-----
Middle Atlantic States.....	221	6	8	15	192	6.3
East North Central States.....	57	-----	4	2	51	7.0
West North Central States.....	29	2	-----	2	25	6.9
South Atlantic States.....	137	14	7	10	106	15.3
East South Central States.....	2	-----	-----	-----	2	-----
West South Central States.....	11	-----	-----	-----	11	-----
Mountain States.....	73	8	9	12	44	23.3
Pacific States.....	140	25	37	18	60	44.3
Hawaii, Canal Zone and Puerto Rico.....	4	-----	1	1	2	-----
Total.....	680	56	67	60	497	18.1

The high proportion of sera showing protection that came from the Pacific and Mountain States is due to the inclusion in table 1 of the 165 sera from these areas collected by us. These were collected after considering some of the peculiarities of an institutional epidemic of central nervous system disease which attacked 198 employees of the Los Angeles County General Hospital coincidentally with an outbreak of poliomyelitis in Los Angeles County in 1934.

In a recent report on this outbreak Gilliam (10) pointed out several unique features not commonly encountered in poliomyelitis epidemics. Some of these were as follows:

(a) An attack rate of 4.4 percent for all hospital employees with nurses and physicians suffering attack rates of 10.7 percent and of 5.4 percent, respectively;

(b) Ninety-two percent of the patients gave a history of direct contact with a prior case within the 21 days before onset of illness;

(c) The peculiar clinical course of the illness with the infrequent occurrence of muscle atrophy; and

(d) The absence of fatal cases among the employees.

After considering these and other peculiarities in this epidemic, a study of some of the cases which developed within as well as without the Los Angeles General Hospital for antibodies capable of neutralizing the virus of lymphocytic choriomeningitis was decided upon.

The subjects were bled under sterile precautions and the sera were separated from the clots and mailed in sterile rubber-stoppered vials to the National Institute of Health for study. Each vial was identified by number only, hence the name of the donor and his clinical history were unknown to those performing the tests (J. G. W. and C. A.).

TABLE 2.—Source of blood sera and the results of the tests for antibodies against the lymphocytic choriomeningitis virus

Source of blood sera	Total number of sera tested	Number of sera showing—				Per cent of sera showing protection
		Strong protection	Moderate protection	Questionable protection	No protection	
(a) Convalescent patients of Los Angeles County Hospital who exhibited central nervous system symptoms*.....	25	5	11	3	6	64.0
(b) Convalescent patients of Los Angeles Orthopedic Hospital who had poliomyelitis during 1934-37.....	8	2	4	1	1	75.0
(c) Employees of the Los Angeles County Hospital who had contact with group (a) patients but who gave no history of central nervous system disease.....	26	7	11	3	5	69.2
(d) Healthy individuals living in Los Angeles who had no contact with the hospital patients.....	33	2	3	2	26	15.1
(e) Medical students and laboratory workers living in San Francisco, Calif.....	21	1	2	6	12	14.2
(f) Laboratory and office workers living in Helena, Mont.....	15	.....	5	4	6	33.3
(g) Laboratory and office workers living in Salt Lake City, Utah.....	17	7	4	5	1	64.7
(h) Medical students, nurses, and laboratory workers living in Portland, Oreg.....	20	8	5	2	5	65.0
Total.....	165	32	45	26	57	46.6
(i) Individuals living in other localities of the United States tested concurrently with the above sera.....	280	10	13	20	237	8.2
Grand total.....	445	42	58	46	294	.....

\*A division of this group into paralytic and nonparalytic cases demonstrated practically the same percentages of individuals that exhibited neutralizing antibodies within each group.

In table 2 are shown the groups of persons from whom sera were obtained together with the results secured in the mouse protection tests. All tests recorded in table 2 are included in table 1. The difference between the total numbers shown in the two tables arises from the fact that 235 sera were tested before the study of the Los Angeles sera was begun. Moreover, 280 blood sera received from miscellaneous locations in the United States were tested concurrently with the 165 sera of interest in connection with the Los Angeles outbreak.

Table 2 shows a high proportion of sera with protective antibodies from five groups of persons, namely (a) convalescent patients of the Los Angeles County General Hospital who had suffered from central nervous system symptoms; (b) convalescent patients of the Los Angeles Orthopedic Hospital who had poliomyelitis during 1934 to 1937; (c) employees of the Los Angeles County Hospital who had contact with group (a) patients but who gave no history of central nervous system disease; (j) laboratory and office workers living in

Salt Lake City, Utah; and (h) medical students, nurses, and laboratory workers living in Portland, Oregon.

The sera from noncontact persons in San Francisco and Los Angeles, and in Salt Lake City, Utah, Portland, Oregon, and Helena, Montana, were collected as noncontact controls for the hospital epidemic study. These donors were healthy individuals who could not recall having suffered from any central nervous system disorder nor having had obvious contact with such cases.

At first glance, the high percentage of protection in the sera of the hospital patients might suggest that lymphocytic-choriomeningitis virus was in part responsible for the epidemic and played some part in the symptomatology. There is little evidence to bear out this hypothesis, however, since the sera of only 25 of the 189 persons ill during the epidemic were tested. Also, no sera were collected from these persons during the early part of their disease for comparative study with the convalescent sera here reported. It is evident from these tests that a large proportion of patients of the Los Angeles County Hospital, as well as their contacts, showed the presence of antibodies capable of inactivating the virus of lymphocytic choriomeningitis. While the tests on 25 sera collected from healthy noncontacts living in Los Angeles revealed 15.1 percent which gave some degree of protection, when and where these immune individuals from both groups had contact with the virus is, of course, unknown.

TABLE 3.—Results of the tests for antibodies against encephalitis, St. Louis type, and lymphocytic choriomeningitis viruses (same sera)

Source of sera	Total number of sera tested	Encephalitis virus					Lymphocytic choriomeningitis virus				
		Protection				Percent of sera showing protection	Protection				Percent of sera showing positive
		Strong	Moderate	Questionable	None		Strong	Moderate	Questionable	None	
Patients of Los Angeles hospitals who had central nervous system symptoms.....	12	---	1	3	8	8.3	1	6	3	2	58.3
Employees of the General Hospital who had contact with the hospital patients but had no history of central nervous system disease.....	8	---	---	---	8	---	3	1	1	3	50.0
Healthy individuals living in Los Angeles who had no contact with the hospital patients.....	10	---	---	---	10	---	2	---	2	6	20.0
Healthy persons living in localities distant from Los Angeles.....	11	---	---	---	11	---	1	2	2	6	27.3
Total sera tested.....	41	---	1	3	37	2.7	7	9	8	17	38.0

Tests for antibodies capable of neutralizing the virus of encephalitis, St. Louis type, were made on 41 of the sera studied in table 2. Of these 41 sera, 12 were from patients ill during the hospital epidemic, 8 were from contacts with these patients, 10 were from noncontacts

living in Los Angeles, and 11 were sera taken from the controls living distant from Los Angeles. It is shown in table 3 that only one of these sera gave protection against the encephalitis virus, while protection against the virus of lymphocytic choriomeningitis was found in 16, or 38 percent, of these 41 sera.

#### SUMMARY

1. Antibodies capable of neutralizing the virus of lymphocytic choriomeningitis were found in 123, or 18 percent, of 680 sera tested from various parts of the United States and Hawaii.

2. Antibodies were found in sera from all geographical groups of States of the United States except 2. In these 2 exceptions only 2 and 11 sera, respectively, were tested.

3. Protective antibodies were found in blood sera of 16, or 64.0, percent, of 25 patients convalescent from illness of the central nervous system suffered during an epidemic in the Los Angeles County Hospital in 1934, and in 6 of 8 convalescents from the Los Angeles Orthopedic Hospital ill from 1934-37, and in the sera of 18, or 69.2 percent, of 26 employees of the Los Angeles County Hospital who had come in contact with the 1934 outbreak.

4. Antibodies were found in the sera of 11 of 17 laboratory and office workers living in Salt Lake City, Utah, and in 13 of 20 medical students, nurses, and laboratory workers of Portland, Oreg.

5. The virus of encephalitis, St. Louis type, was neutralized by only one of 41 sera tested, while 16 of the same sera neutralized the virus of lymphocytic choriomeningitis.

6. Results of the serum-virus protection test indicate that the virus of lymphocytic choriomeningitis is widely spread in the United States.

7. Individual histories of cases possessing virus-neutralizing antibodies in their blood indicate that infection with the virus was not identified as to etiology, in many cases, or was possibly asymptomatic.

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## ACUTE RESPONSE OF GUINEA PIGS TO THE INHALATION OF DIMETHYL KETONE (ACETONE) VAPOR IN AIR<sup>1</sup>

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The effects of acetone administration have been rather extensively investigated in the various laboratory animals, mice, rats, rabbits, and dogs (1). The data recorded in each case were mainly intended to furnish an index of lethal or narcotic dose, and to ascertain the presence or absence of recognizable injury due to chronic or acute exposure. The course of the intoxication was followed, in most cases, to record time of occurrence of overt symptoms at stated dosages, but these data were not presented in a correlated manner. It is our purpose to present quantitative data on the physiological effects of acetone inhalation in guinea pigs in the same manner that the data on methyl isobutyl ketone (2) were presented.

### USES

Acetone is commonly used as a solvent for resins, lacquers, fats, oils, collodion cotton, celluloid, cellulose acetate, and acetylene.

### PHYSICAL PROPERTIES

Acetone is a volatile, inflammable, colorless liquid, with a specific gravity of 0.792 at 20° C. It boils at 56.5° C. and has a vapor pressure of 235 mm. Hg at 25° C. It is miscible with water in all proportions.

Explosive limits at room temperature are given by Piatti (3) as 2.5 to 9.0 volumes percent.

The acetone used in these experiments was of technical grade and boiled at 55.5-56.2° C. It was found to be free from methanol.

<sup>1</sup> From the Division of Industrial Hygiene, National Institute of Health. Read before the American Society for Pharmacology and Experimental Therapeutics at the annual meeting of the Federation of American Societies of Experimental Biology in Toronto, Canada, April 26-29, 1939.

## ANALYSIS

The concentration of acetone vapor in the exposure chamber was determined by the iodine titration method, using the precautions recommended by Goodwin (4). The procedure followed was that used by Patty, Schrenk, and Yant (5) and modified by Reinhart (2). According to Haughton (6) the apparent recovery by this method is 102.5 percent.

## APPARATUS

The exposures were made in a glass-walled box described in our first paper (2) but modified in several details. Since temperature regulation was found advisable, as the season progressed, a circulating blower circuit was installed. This circuit is made of 2-inch spouting and contains a copper coil for adding or removing heat, respectively, to or from the vapor-air mixture forced past it. Regulation is accomplished by the use of a mercury regulator at the exit part of the circuit, operating a solenoid water valve through an appropriate relay system.

For the measurement of rectal temperature, it was found expedient to use a thermocouple arrangement. The standard couple is immersed in water in a Dewar flask with a sensitive thermometer and the water temperature is read each time a series of rectal temperatures is taken. The galvanometer deflection is read in arbitrary units and converted to degrees after correcting for the change in temperature of the standard couple. In this case the standard couple was kept at approximately 32° C. This means of temperature measurement has several advantages over the usual methods: There is a relatively small mass which must be heated by the body, thus insuring an equilibrium temperature relatively near the true temperature; the reading can be made in about 30 seconds; and there is no necessity to make any adjustments between readings such as in shaking down a clinical thermometer.

The respiratory rate was measured with a stop watch by direct observation. The type of respiration was noted in each case. Quantitative methods of recording the respiratory rate and volume by mechanical displacement could not be fitted satisfactorily enough to warrant the inclusion of the results which were obtained.

The heart rate was estimated in an arbitrary manner with good results by using two observers—one to detect the rate by palpation and match a short repetitive verbal count (1 to 10) to this rate and the other to time a convenient group of counts with the stop watch.

Considerable experimentation in the method of maintaining the vapor concentration in the absence of a flowing atmosphere resulted in the construction of an evaporating surface made of four filter papers

folded and clipped together in such a fashion as to make an hour-glass-shaped device which hangs under the inlet of the circulating blower. Ketone is added as needed from a burette outside the chamber by means of a glass tube leading to a point over the evaporator.

CONTROLS

From the control runs it appears that the humidity reached only 70 percent even after nearly 14 hours of confinement. The carbon dioxide concentration reached less than 4 volumes percent in this period. In view of these data, it does not seem that significant physiological changes were produced in the presence of the various concentrations of ketone by the accumulation of water vapor and carbon dioxide, or the lack of oxygen. Figure 1 shows the course of events during repeated control runs. The plotted points are averages of data from 10 guinea pigs. The lines drawn through them indicate

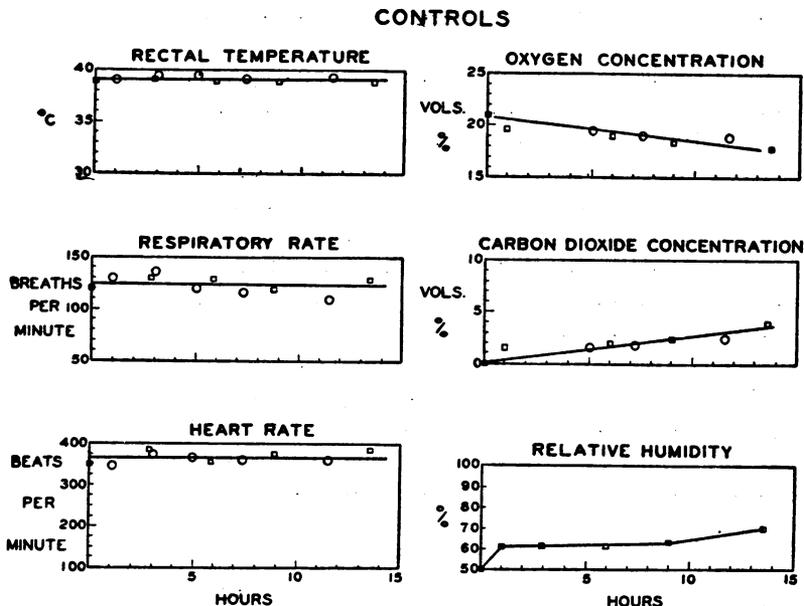


FIGURE 1.—Control experiments. Ten guinea pigs in the 1 cubic meter chamber, sealed, and temperature controlled within 0.5° C. at 25° C. Square and round points denote average values of two different runs. Lines through the points of rectal temperature, respiratory rate, and heart rate indicate general average; others follow trend of the points.

the general average and are intended only to bring out the trend of the data.

METHOD

The exposures were made at 25° ± 1° C. Data were collected at 1, 2, and 5 volumes percent at progressively longer periods throughout the duration of the experiments with particular regard to the following plan in which are arranged the responses or symptoms of guinea pigs in order of increasing severity of reaction:

1. Irritation of cornea and conjunctiva (squinting, lacrimation, rubbing of eyes).
2. Irritation of buccal, nasal, and pharyngeal passages (sneezing, coughing, salivation, retching, rubbing of nose and mouth).
3. Narcotization of central nervous system (postural instability, loss of auditory and corneal reflexes, change in respiratory rate and character, coma and death).
4. Changes in metabolism of the tissues (changes in rectal temperature, change in pulse, etc.).

Air samples were drawn from the chamber at frequent intervals to ascertain the concentration of acetone. If the concentration was found to be low, more acetone was added.

Autopsies were made on pigs that died during the exposure and on some survivors.

#### RESULTS

It is obvious that the only data listed above which lend themselves to a quantitative treatment are respiration, temperature, and pulse. These are presented in some detail by means of three-dimensional charts in which each experiment (at a different concentration of acetone) is represented by a plane whose upper edge is limited by the average of the observed values throughout the duration of the exposure (figs. 2, 3, and 4). In order to have each curve start from the same initial value, the observed values for each concentration were multiplied by a factor. This factor was obtained by dividing the average initial value by the observed initial value at each concentration.

The qualitative data have been used to build up a chart, comparable with those published by Patty, Schrenk, and Yant (5), in which four general conditions are presented in order of their appearance: Irritation, weakness, coma, and death (fig. 5). The lines drawn on the graph represent only our interpretation of the general course of the data, since complete reaction of the whole sample of animals did not occur in many cases.

The general purpose of this section can be best served by recapitulating the protocol of an exposure at an average concentration of 2 volumes percent. A comparison with effects at the other concentrations follows this protocol.

Two liters of acetone were evaporated in the large and small chamber and a concentration of 2.18 volumes percent was measured after the introduction of the animals. The chamber temperature in these early experiments was not controlled but maintained itself at  $26.5^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$ . Ten female guinea pigs of approximately 500 grams body weight were placed in the chamber via the air lock. Their normal rectal temperature, respiratory rate, and pulse were, respectively,  $38.4^{\circ}\text{C}$ ., 108 per minute, and 350 per minute. All values given here are averages of survivors.

*Up to 25 minutes.*—Slight lacrimation, no signs of salivation, no rubbing. two pigs squinting.

*240 minutes.*—One pig has lost auditory response (1), others not very steady on their feet, somewhat restless, fur standing on end, very slight signs of lacrimation, no squint, rectal temperature, 36.7° C.; respiration, 76 per minute, pulse, 297 per minute; concentration of acetone, 1.99 volumes percent.

*505 minutes.*—All but two pigs are so weak they cannot stand, two are without auditory reflex, and two others are unconscious. Concentration of acetone, 1.92 volumes percent.

*540 minutes.*—All pigs but one are in coma, all but two have auditory reflex, all have positive corneal reflex; the effort to right the body when it is rotated by hand (righting reflex) is now very poor. Despite this comatose state there is occasional movement as of rousing for a few seconds and a good deal of pawing or walking movement. Lacrimation is still not marked, but four pigs are beginning to salivate. Rectal temperature, 33.8° C.; respiratory rate, 76 per minute; pulse, 273 per minute.

*865 minutes.*—All on side in coma, all but two pigs with positive auditory reflex, two have lost corneal reflex, only one shows any semblance of righting reflex, all fairly quiet, six are lacrimating, and seven are salivating. Rectal temperature, 31.2° C.; respiratory rate, 64 per minute; pulse, 269 per minute.

*1,340 minutes.*—All auditory reflexes are gone, three pigs are dead, no further lacrimation or salivation, no righting reflex, no corneal reflex.

*1,355 minutes.*—Fourth death.

*1,365 minutes.*—Fifth death. Rectal temperature, 26.7° C.; respiratory rate, 47 per minute; pulse, 214 per minute.

*1,380 minutes.*—Sixth and seventh deaths.

*1,405 minutes.*—Three survivors removed from cage. All pigs were observed to be in rigor even before death. Two pigs were exsanguinated and the other pig died after several hours.

In this and other exposures it was observed, while handling the pigs during the later hours of the exposure, that they were somewhat rigid in the posterior portion of the body. This condition persisted for at least a day in the survivors, and acetone could be detected in their breath for several days subsequent to the exposure. The condition is not so much a spastic paralysis as an exaggerated stiffness similar to acid rigor. We have found no reference to this condition in the literature on acetone, although several references were made to "paralysis." On close scrutiny of the context the condition was interpreted as flaccid paralysis in every case.

#### PATHOLOGY

*Exposure 1 percent.*—Five guinea pigs were autopsied. They had received between 47 and 48 hours' exposure to 1 percent acetone vapor by volume in air. Of them, two died during exposure, a third at the end of 48 hours, and the remaining two 24 and 72 hours, respectively, after the end of the exposure. In the liver there were a few scattered cells containing, usually, few fine and medium-sized fat droplets. Their number appeared to bear no relation to the length of exposure or duration of life after exposure. The spleen showed

slight to moderate congestion of the cavernous veins. The lungs showed congestion of interalveolar capillaries, moderate in the two animals dying during exposure, marked in the one dying at the end of the exposure, and least in the one which lived for 72 hours following completion of exposure. Extravasation of red cells into the alveolar spaces, both with and without serum, accompanied the congestion. Slight to moderately-marked perivascular, lymphocytic infiltration occurred in three of the animals. In the kidneys, the glomerular capsules showed a slight to marked distention, while the straight collecting tubules in the medullary rays were dilated to a somewhat lesser degree. Slight congestion of interstitial capillaries occurred. The adrenals, stomach, pancreas, and heart showed no changes of note.

*Exposure 2 percent.*—Nine guinea pigs were autopsied. They had received between 22 and 26 hours' exposure to 2 percent acetone vapor by volume. All died during exposure except one, which was killed immediately afterward. The liver showed the same picture as with exposure to 1 percent. The spleen showed more marked congestion of the cavernous veins and in every case a slight to moderate perifollicular hemorrhage was noted. Areas of hemorrhage in the pulp were present in all of the animals. The lungs showed about the same degree of congestion of interalveolar capillaries as was noted in the 1-percent series. Hemorrhage into the alveolar spaces was essentially the same but there was less serum in the alveoli. Moderate to marked perivascular lymphoid infiltration occurred in most of the animals. In the kidney, distention of glomerular capsules was practically the same but dilatation of the straight collecting tubules was not as frequent. Slight to moderate congestion of the interstitial capillaries and glomeruli occurred in all but two of the animals. The adrenals, stomach, and pancreas showed no histopathological changes.

*Exposure 5 percent.*—Eight guinea pigs were autopsied. They had received between 3 and 4 hours' exposure to a concentration of 5 percent acetone vapor. All died during exposure. In the liver, small to moderate amounts of fat in the form of large, medium, and small droplets were found in all of the animals. It was mostly in the subcapsular zone and appeared to be of no pathological significance.

In the spleen, moderate to marked congestion of the cavernous veins and slight to moderate hemorrhage in the pulp and about the follicles occurred in all animals. This was more marked than in the 2-percent series. Pulmonary congestion and edema were likewise more prominent and in many of the animals were present to a marked degree. In the kidney, slight to moderate congestion of the capillaries and glomeruli was noted, but the glomerular capsules were no longer distended. The adrenals, stomach, and pancreas showed no changes.

Another series of six guinea pigs was autopsied. They had received

7½ to 8½ hours' exposure to 5 percent acetone vapor. Two died during exposure and the remaining four died from 1½ to 13 hours after conclusion of the experiment. The findings here were essentially the same as in the previous 5-percent series, except for a slight to moderate distention of the glomerular capsules in the kidneys.

A group of four guinea pigs was autopsied. They had received 6½ hours' exposure to 5 percent vapor. Two were killed and the other two died 13 hours later. With the exception of the lungs (death was effected by exsanguination), the pathological picture was the same.

RECTAL TEMPERATURE OF GUINEA PIGS IN ACETONE VAPOR

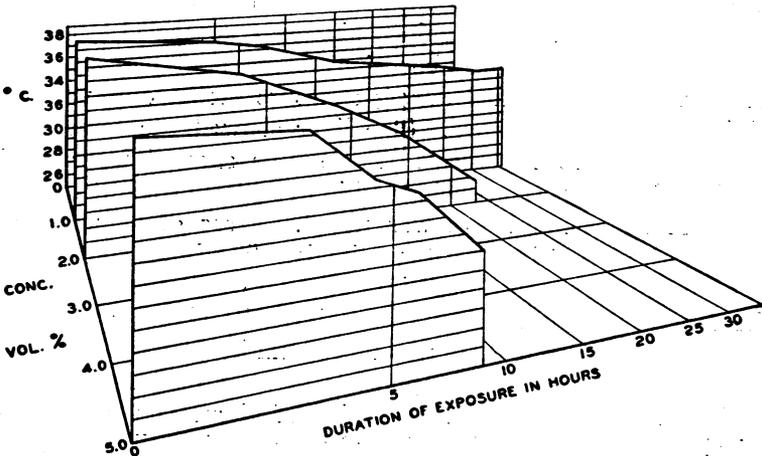


FIGURE 2.—Rectal temperature during exposure to 1.0, 2.0, and 5.0 volumes percent acetone. The ordinates indicate average rectal temperature of at least 10 guinea pigs with the chamber temperature held at 25°±1° C. The abscissa to the left indicates the concentration of acetone in volumes percent and that to the right time in hours, marked at 5-hour periods. Note that the 5.0 and 2.0 volume percent planes end within the grid. This indicates death of the sample; usually all the pigs succumb within a short period following this reading. The last vertical plane represents the average control level.

DISCUSSION

Figure 2 indicates that there is a regular relationship between concentration and rectal temperature. There is shown an increasingly strong effect with concentration as the duration of the exposure increases, until death ensues. The exposure to 1 percent acetone was continued for 13 hours beyond the last point shown in figures 2, 3, and 4, but no significant change in course was observable.

Similarly, in figure 3, the respiratory rate falls off with time at an increasing rate with increasing concentration; however, it is to be noted that with 5 percent acetone there is an immediate checking of respiration, which is mainly the result of irritation of the mucosa of the nose and pharynx. Later, as narcosis becomes deeper, the gradual decline of the respiratory rate becomes evident.

## RESPIRATORY RATE OF GUINEA PIGS IN ACETONE VAPOR

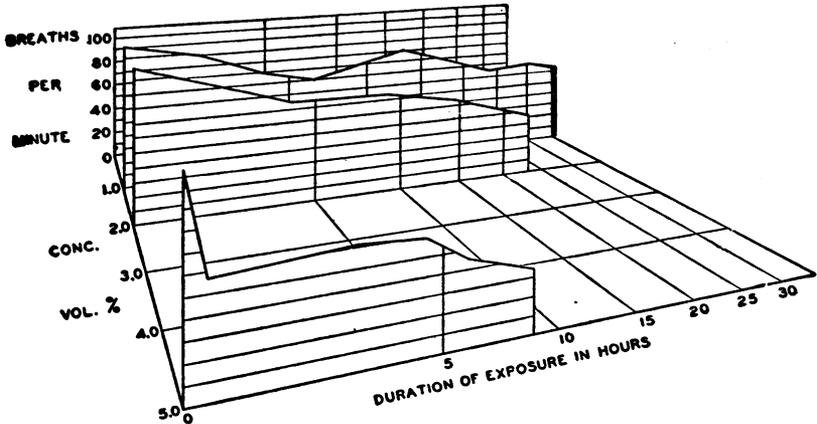


FIGURE 3.—Respiratory rate during exposure to 1.0, 2.0, and 5.0 volumes percent acetone. The ordinates indicate the average respiratory rates of at least 10 guinea pigs with the chamber temperature maintained at  $25^{\circ}\pm 1^{\circ}$  C. The abscissa to the left indicates the concentration of acetone in volumes percent and that to the right time in hours, marked at 5-hour intervals. Note that the 5.0 and 2.0 volume percent planes end within the grid. This indicates death of the sample; usually all the pigs succumb shortly after this reading. The last vertical plane represents the average control level.

## HEART RATE OF GUINEA PIGS IN ACETONE VAPOR

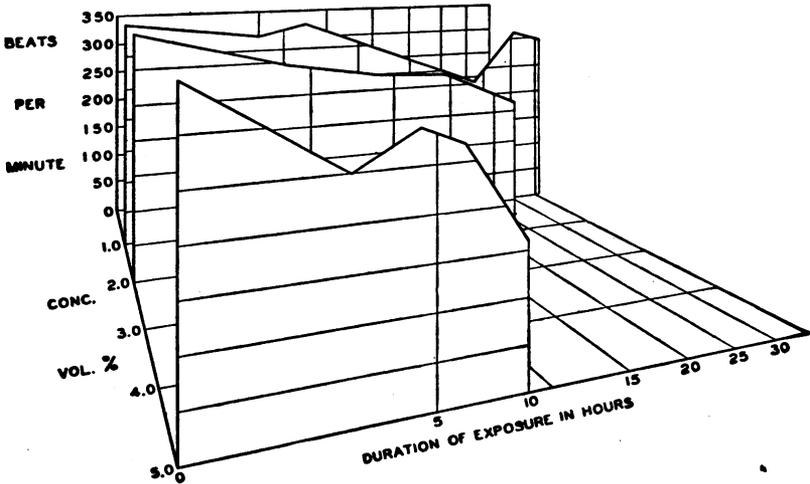


FIGURE 4.—Heart rate during exposure to 1.0, 2.0, and 5.0 volumes percent acetone. The ordinates indicate the average respiratory rates of at least 10 guinea pigs, with the chamber temperature maintained at  $25^{\circ}\pm 1^{\circ}$  C. The abscissa to the left indicates the concentration of acetone in volumes percent and that to the right time in hours, marked at 5-hour intervals. Note that the 5.0 and 2.0 volume percent planes end within the grid. This indicates death of the sample; usually all the pigs succumb shortly after this reading. The last vertical plane represents the average control level.

Figure 4 gives similar data on heart rate. Despite the obvious irregularity, the trend is very similar and serves to corroborate the general nature of the narcosis which acetone produces. We have found that changes in the regular fall in heart rate and respiratory rate under exposure vary with the time of day in these long exposures.

A study of blood acetone was begun during the exposures to 1, 2, and 5 volumes percent acetone in the air described in this paper. The data, though incomplete,<sup>2</sup> show that acetone is not removed rapidly

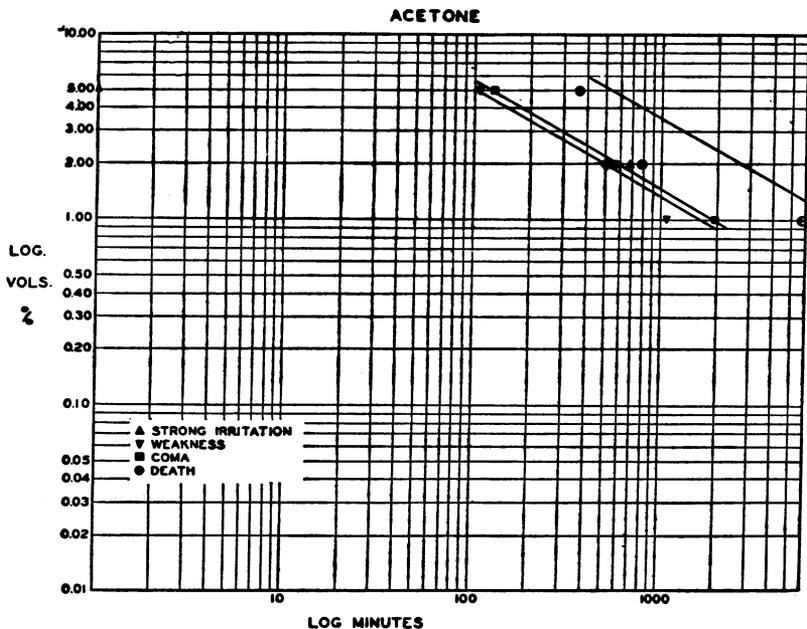


FIGURE 5.—Concentration versus time chart of symptoms of acetone intoxication. The average time of reaction of the whole sample is shown by the filled points, reaction of part of the sample by partly filled points. The ordinate represents the logarithm of the concentration of acetone in volumes percent and the abscissa the logarithm of time in minutes. Note that in the experiment at 2.0 volumes percent acetone the time of irritation falls after coma. This was judged mainly on the basis of lacrimation and salivation, which did appear subsequent to the narcosis.

enough from the blood to maintain an equilibrium between the blood and inspired air at these levels.

One volume percent of acetone in the inhaled air is strongly irritating to man and would not be tolerated under any but exceptional conditions. Gradually mounting concentrations may, however, dull the sensation of irritation and raise the threshold of intolerable concentrations to such a point where accumulation may take place. The capacity for accumulation is indicated by the above findings.

From the literature examined and reprinted by Browning (1) it is evident that the general objective symptoms are narcosis and spasmodic movement in the higher concentrations. By referring to figure

<sup>2</sup> Blood changes in acetone intoxication are now being investigated.

5 it can be seen that the objective symptoms in our experiments corroborate the narcosis but illustrate a remarkable tolerance for acetone as compared with other ketones (2).

The observations of Salant and Kleitman (7) and Walton et al. (8) on the effect of acetone in reducing cardiac output, and the observations of Gollwitzer-Meier (9), Tappeimer (10) and Schlomowitz and Seybold (11) on the respiratory depression are corroborated generally by our data on respiratory rate and heart rate. The decline of these reactions is shown to be of a continuous nature in our own findings.

The authors cited above find an initial period of stimulation in acetone intoxication. In our opinion, under light narcosis there is blocking of inhibitions accompanied by a seeming exhilaration which may appear to be the result of stimulation. We have regularly observed this restless period before deep coma sets in.

Under the conditions of these experiments, there is no indication that any stimulation or increase occurs in heart rate or respiratory rate such as would result from anoxemia.

#### SUMMARY

1. Inhalations of 1, 2, and 5 percent acetone vapor in air were studied in respect to change in rectal temperature, respiration, and pulse of guinea pigs. Symptomatic data of a qualitative nature were also recorded.

2. As the duration of exposure increases there is a gradual fall in rectal temperature, respiratory rate, and pulse. The rate of this change is roughly proportional to the concentration of acetone vapor.

3. In general, as the concentration rises, the effect at any stated interval becomes more marked.

4. It is concluded from qualitative and quantitative data that there is a progressive general narcosis.

5. The concentration of acetone in the blood is progressively greater as the exposure time increases. No equilibrium was reached and saturation values were not attained.

6. In animals exposed to 1 percent of acetone vapor for approximately 48 hours, congestion of the spleen, congestion and edema of the lungs, and distention of glomerular capsules and the straight collecting tubules of the kidney occurred.

7. In animals exposed to 2 percent vapor for 22 to 26 hours similar changes were noted. The congestion of the cavernous veins of the spleen were more marked and perifollicular and interstitial hemorrhage was noted. Pulmonary congestion was approximately the same. The distention of glomerular capsules in the kidney was essentially un-

changed, but the distention of the straight tubules was not as frequent. Renal congestion was present.

8. In animals exposed to 5 percent vapor for 3 to 8¼ hours, pulmonary congestion and edema and splenic congestion and hemorrhage were more marked. In the animals receiving the longer exposure and which died from 1½ to 13 hours after completion of exposure, distention of renal glomerular capsules was noted, but in the animals which died during shorter exposures only renal congestion was noted.

It appears that the pulmonary congestion and edema are due to the direct irritation of the vapor. The splenic damage is most marked in the short exposures to higher concentrations. Congestion of the renal capillaries and glomeruli likewise is present under these conditions but distention of the tubules and glomerular capsules occurs with long exposures to low concentrations.

#### ACKNOWLEDGMENTS

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## NEW MOSQUITO RECORDS FROM KEY WEST, FLA.

Junior Entomologist Frank W. Fisk, of the United States Public Health Service, has recently reported <sup>1</sup> that *Culex bahamensis* Dyar and Knab, a species of mosquito heretofore known only on certain islands of the West Indies, has been found to be fairly common at Key West, Fla., by the *Aedes aegypti* Control Unit of the Public Health Service. Larvae of this species were first found in an abandoned brackish cistern, in company with *Anopheles atropos*, on January 24, 1939. Since then other breeding places have been located and adults of the species have been taken regularly with light traps. Extensive surveys in Miami and environs have failed to show the presence of *Culex bahamensis* there.

The discovery of *Anopheles atropos* is interesting, as it is reported to be the first anopheline recorded from Key West since the discovery and apparent eradication of *Anopheles albimanus* Wied. from the island in 1904.

## DEATHS DURING WEEK ENDED MAY 13, 1939

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended May 13, 1939	Correspond- ing week, 1938
<b>Data from 88 large cities of the United States:</b>		
Total deaths.....	8,612	<sup>1</sup> 7,976
Average for 3 prior years.....	<sup>2</sup> 8,297	
Total deaths, first 19 weeks of year.....	175,371	166,990
Deaths under 1 year of age.....	518	<sup>1</sup> 517
Average for 3 prior years.....	<sup>2</sup> 530	
Deaths under 1 year of age, first 19 weeks of year.....	10,244	10,293
<b>Data from industrial insurance companies:</b>		
Policies in force.....	67,406,340	68,329,739
Number of death claims.....	15,187	12,494
Death claims per 1,000 policies in force, annual rate.....	11.7	9.5
Death claims per 1,000 policies, first 19 weeks of year, annual rate.....	11.7	10.0

<sup>1</sup> Data for 87 cities.

<sup>2</sup> Data for 86 cities.

<sup>1</sup> Journal of Economic Entomology, June 1939.

# PREVALENCE OF DISEASE

*No health department, State or local can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring*

## UNITED STATES

### CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (.....) represent no report, with the implication that cases of deaths may have occurred but were not reported to the State health officer.

*Cases of certain diseases reported by telegraph by State health officers for the week ended May 20, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median*

Division and State	Diphtheria				Influenza				Measles			
	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median
<b>NEW ENG.</b>												
Maine.....	6	1	1	1	84	14	1	1	935	155	103	103
New Hampshire.....	0	0	0	0	.....	.....	.....	.....	30	3	42	35
Vermont.....	0	0	0	0	.....	.....	.....	.....	1,113	82	138	65
Massachusetts.....	8	7	1	6	.....	.....	.....	.....	1,475	1,254	272	716
Rhode Island.....	8	1	0	0	.....	.....	.....	.....	611	80	1	68
Connecticut.....	6	2	7	2	9	3	3	2	2,469	832	53	189
<b>MID. ATL.</b>												
New York.....	10	25	25	37	16	19	17	17	901	2,251	3,351	3,876
New Jersey.....	12	10	7	15	8	7	3	7	80	67	845	845
Pennsylvania.....	11	22	29	29	.....	.....	.....	.....	70	138	4,597	3,438
<b>E. NO. CEN.</b>												
Ohio.....	4	5	6	12	.....	.....	.....	24	15	19	1,114	1,114
Indiana.....	10	7	35	10	49	33	5	12	16	11	462	462
Illinois <sup>1</sup> .....	14	22	25	37	11	17	16	29	35	54	1,319	1,319
Michigan <sup>2</sup> .....	8	8	15	14	4	4	.....	.....	590	564	3,140	322
Wisconsin.....	2	1	4	2	84	48	8	53	1,434	816	2,585	1,505
<b>W. NO. CEN.</b>												
Minnesota.....	4	2	1	2	6	3	.....	1	516	266	370	370
Iowa <sup>1</sup> .....	4	2	6	6	2	1	.....	.....	308	152	290	290
Missouri.....	6	5	19	19	.....	9	36	9	7	198	198	198
North Dakota.....	0	0	0	1	592	81	3	3	409	56	96	15
South Dakota.....	8	1	0	0	15	2	.....	.....	1,803	240	0	5
Nebraska.....	4	1	1	1	.....	.....	.....	.....	813	213	234	234
Kansas.....	11	4	5	7	8	3	9	4	221	79	407	407
<b>SO. ATL.</b>												
Delaware.....	20	1	3	1	.....	.....	.....	.....	216	11	14	24
Maryland <sup>1,2</sup> .....	3	1	4	5	9	3	3	3	981	318	55	408
Dist. of Col.....	8	1	3	10	.....	.....	.....	.....	3,161	391	14	75
Virginia <sup>1</sup> .....	11	6	12	12	201	107	.....	.....	1,614	861	413	502
West Virginia.....	13	5	4	6	70	26	24	20	5	2	267	154
North Carolina.....	9	6	11	15	.....	.....	3	5	690	472	1,695	272
South Carolina <sup>1</sup> .....	19	7	2	2	1,000	366	47	72	30	11	68	82
Georgia <sup>1</sup> .....	7	4	9	5	247	149	.....	.....	219	132	154	0
Florida <sup>1</sup> .....	3	1	1	3	48	16	2	3	292	97	78	26

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended May 20, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median
<b>E. SO. CEN.</b>												
Kentucky	17	10	6	6	16	9	1	8	63	36	158	283
Tennessee	4	2	2	4	102	58	24	42	101	57	113	113
Alabama	14	8	16	5	331	188	17	29	310	176	804	122
Mississippi	13	5	5	5								
<b>W. SO. CEN.</b>												
Arkansas	10	4	5	5	72	29	23	26	352	142	184	54
Louisiana	31	13	5	12	10	4	14	6	162	67	37	37
Oklahoma	12	6	3	5	121	60	51	51	451	224	164	86
Texas	19	23	34	37	340	410	176	176	358	432	260	325
<b>MOUNTAIN</b>												
Montana	75	8	2	2	646	69		21	1,432	153	62	62
Idaho	0	0	0	0				7	1,337	131	23	23
Wyoming	0	0	0	0					1,811	83	26	23
Colorado	39	8	6	6	19	4			1,141	237	319	319
New Mexico	0	0	1	1	12	1	12	9	124	10	114	82
Arizona	0	0	4	1	577	47	27	25	159	13	18	18
Utah	0	0	0	0	30	3			1,500	151	317	52
<b>PACIFIC</b>												
Washington	0	0	0	1					3,290	1,067	28	132
Oregon	15	3	1	2	174	35	27	19	388	78	58	75
California	29	35	26	26	33	40	44	44	2,061	2,513	978	978
Total	11	283	353	396	87	1,849	666	789	614	15,205	25,538	25,538
20 weeks	17	8,751	10,388	10,966	343	145,395	40,899	99,537	553	273,815	638,672	533,467

Division and State	Meningitis, meningococcus				Pollomyelitis				Scarlet fever			
	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median
<b>NEW ENG.</b>												
Maine	0	0	0	0	0	0	0	0	66	11	26	21
New Hampshire	0	0	0	0	0	0	0	0	41	4	21	15
Vermont	0	0	0	0	0	0	0	0	67	5	8	8
Massachusetts	5	4	1	1	0	0	0	0	180	153	398	259
Rhode Island	8	1	0	0	0	0	0	0	31	4	15	19
Connecticut	3	1	0	1	0	0	0	0	157	53	86	86
<b>MID. ATL.</b>												
New York	2	5	8	10	0	0	1	1	222	554	714	781
New Jersey	0	0	2	2	0	0	0	0	273	229	101	181
Pennsylvania	2	4	5	5	0	0	0	1	150	295	665	598
<b>E. NO. CEN.</b>												
Ohio	0	0	1	6	0	0	0	0	211	274	202	320
Indiana	1.5	1	0	2	0	0	0	0	204	137	76	96
Illinois	0	0	4	4	1.3	2	1	1	258	394	402	570
Michigan	0	0	0	0	0	0	2	1	486	460	384	384
Wisconsin	1.8	1	1	1	0	0	0	0	225	128	139	431

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended May 20, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued.

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median
<b>W. NO. CEN.</b>												
Minnesota.....	0	0	0	0	0	0	0	0	147	76	127	137
Iowa <sup>1</sup> .....	0	0	1	2	0	0	0	0	156	77	96	96
Missouri.....	1.3	1	0	4	0	0	0	0	87	68	127	127
North Dakota.....	0	0	0	0	0	0	0	0	124	17	24	39
South Dakota.....	0	0	0	0	0	0	0	0	83	11	12	25
Nebraska.....	0	0	0	2	0	0	0	0	95	25	24	56
Kansas.....	2.8	1	1	1	2.8	1	0	0	131	47	98	98
<b>SO. ATL.</b>												
Delaware.....	0	0	0	0	0	0	0	0	118	6	10	3
Maryland <sup>1,2</sup> .....	0	0	2	2	0	0	0	0	93	30	78	50
Dist. of Col.....	0	0	1	2	0	0	0	0	97	12	14	17
Virginia <sup>1</sup> .....	0	0	3	5	0	0	1	1	34	18	17	17
West Virginia.....	2.7	1	2	4	0	0	0	0	67	25	35	62
North Carolina.....	0	0	1	2	0	0	1	1	15	10	17	7
South Carolina <sup>1</sup> .....	2.7	1	0	0	76	28	0	0	8	3	0	2
Georgia <sup>1</sup> .....	0	0	1	1	0	0	0	0	22	13	16	14
Florida <sup>1</sup> .....	0	0	0	0	3	1	3	0	30	10	4	4
<b>E. SO. CEN.</b>												
Kentucky.....	1.7	1	9	9	0	0	0	0	82	47	25	25
Tennessee.....	0	0	1	5	1.8	1	0	0	101	57	21	16
Alabama <sup>1</sup> .....	0	0	0	1	0	0	1	0	11	6	3	5
Mississippi <sup>1</sup> .....	0	0	1	1	0	0	0	0	8	3	5	5
<b>W. SO. CEN.</b>												
Arkansas.....	7	3	0	0	5	2	0	0	25	10	4	4
Louisiana <sup>1</sup> .....	0	0	0	1	2.4	1	2	1	39	16	7	7
Oklahoma.....	0	0	1	1	0	0	0	0	32	16	14	14
Texas <sup>1</sup> .....	2.5	3	1	3	2.5	3	1	1	27	32	74	46
<b>MOUNTAIN</b>												
Montana <sup>1</sup> .....	0	0	0	0	0	0	0	0	112	12	11	11
Idaho <sup>1</sup> .....	10	1	0	0	0	0	0	0	20	2	5	8
Wyoming <sup>1</sup> .....	0	0	0	0	0	0	0	0	109	5	2	15
Colorado <sup>1,2</sup> .....	10	2	0	0	0	0	0	0	144	30	68	58
New Mexico.....	12	1	1	0	0	0	0	0	86	7	6	14
Arizona.....	0	0	0	0	0	0	0	0	147	12	7	10
Utah <sup>1,2</sup> .....	0	0	0	0	0	0	0	0	298	30	15	16
<b>PACIFIC</b>												
Washington <sup>1</sup> .....	0	0	0	1	0	0	0	0	173	56	25	48
Oregon <sup>1</sup> .....	0	0	0	0	0	0	0	0	89	18	22	25
California.....	1.6	2	2	4	2.5	3	2	3	134	164	166	210
Total.....	1.4	34	50	102	1.7	42	15	19	146	3,672	4,406	5,616
20 weeks.....	1.9	968	1,592	2,843	0.8	413	390	412	195	97,895	113,890	134,892

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended May 20, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued.

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases	1934-38, median	May 20, 1939, rate	May 20, 1939, cases	May 21, 1938, cases
<b>NEW ENG.</b>											
Maine.....	0	0	0	0	6	1	0	1	290	48	55
New Hampshire.....	0	0	0	0	0	0	1	0	41	4	0
Vermont.....	0	0	0	0	0	0	4	1	295	22	73
Massachusetts.....	0	0	0	0	2	1	2	2	140	119	115
Rhode Island.....	0	0	0	0	8	1	0	0	657	86	21
Connecticut.....	0	0	0	0	3	1	1	1	122	41	97
<b>MID. ATL.</b>											
New York.....	0	0	0	0	1	3	5	8	164	409	463
New Jersey.....	0	0	0	0	4	3	4	2	327	275	---
Pennsylvania.....	0	0	0	0	2	4	13	5	151	297	254
<b>E. NO. CEN.</b>											
Ohio.....	2	3	5	0	8	10	5	5	55	71	141
Indiana.....	43	29	39	3	4	3	3	3	65	44	21
Illinois <sup>1</sup> .....	11	17	17	13	4	6	7	6	130	198	141
Michigan <sup>2</sup> .....	4	4	7	2	4	4	2	3	165	156	255
Wisconsin.....	5	3	3	7	4	2	0	1	299	170	208
<b>W. NO. CEN.</b>											
Minnesota.....	16	8	10	7	2	1	0	0	52	27	22
Iowa <sup>3</sup> .....	53	26	28	20	2	1	1	1	53	26	34
Missouri.....	26	29	24	24	0	0	0	3	21	16	23
North Dakota.....	15	2	14	3	0	0	1	0	117	16	23
South Dakota.....	120	16	11	4	0	0	0	0	15	4	16
Nebraska.....	19	5	6	6	0	0	0	0	164	43	16
Kansas.....	17	6	9	9	3	1	0	3	89	32	139
<b>SO. ATL.</b>											
Delaware.....	0	0	0	0	20	1	0	0	157	8	6
Maryland <sup>4</sup> .....	0	0	0	0	6	2	5	5	89	29	62
Dist. of Col.....	0	0	0	0	0	0	0	0	121	15	3
Virginia <sup>5</sup> .....	0	0	0	0	9	5	5	5	54	45	117
West Virginia.....	0	0	1	1	0	0	5	5	56	21	32
North Carolina.....	0	0	3	0	12	8	6	3	318	218	345
South Carolina <sup>6</sup> .....	0	0	0	0	8	3	2	5	287	105	28
Georgia <sup>7</sup> .....	3	2	0	0	10	6	10	11	129	78	91
Florida <sup>8</sup> .....	0	0	0	0	39	13	3	5	172	57	19
<b>E. SO. CEN.</b>											
Kentucky.....	2	1	1	0	17	10	3	5	24	14	84
Tennessee.....	12	7	3	0	4	2	4	4	39	22	56
Alabama <sup>9</sup> .....	0	0	9	0	19	11	2	6	114	65	54
Mississippi <sup>2</sup> .....	3	1	1	0	0	0	9	3	---	---	---
<b>W. SO. CEN.</b>											
Arkansas.....	40	16	6	6	5	2	5	1	79	32	21
Louisiana <sup>4</sup> .....	2	1	0	0	36	15	7	13	102	42	44
Oklahoma.....	66	33	14	4	10	5	8	4	4	2	37
Texas <sup>5</sup> .....	3	4	11	11	6	7	9	14	152	184	364
<b>MOUNTAIN</b>											
Montana <sup>6</sup> .....	0	0	5	8	19	2	1	1	262	28	29
Idaho <sup>7</sup> .....	0	0	10	3	0	0	0	0	71	7	4
Wyoming <sup>8</sup> .....	22	1	0	0	0	0	0	0	44	2	1
Colorado <sup>9</sup> .....	14	3	6	2	5	1	1	0	226	47	38
New Mexico.....	37	3	3	0	0	0	3	2	235	19	7
Arizona.....	61	5	0	0	12	1	7	3	135	11	50
Utah <sup>10</sup> .....	0	0	0	0	20	2	0	0	715	72	45
<b>PACIFIC</b>											
Washington <sup>2</sup> .....	3	1	14	7	0	0	1	1	46	15	174
Oregon <sup>1</sup> .....	40	8	16	8	10	2	3	4	169	34	23
California.....	10	12	70	11	7	9	20	9	232	283	608
Total.....	9	237	354	223	6	150	166	166	144	3,557	4,452
20 weeks.....	14	7,016	10,159	4,442	5	2,360	2,540	2,540	162	80,002	85,571

<sup>1</sup> New York City only.

<sup>2</sup> Rocky Mountain spotted fever, week ended May 20, 1939, 22 cases as follows: Illinois, 1; Iowa, 1; Maryland, 3; Virginia, 1; Montana, 4; Idaho, 2; Wyoming, 5; Colorado, 1; Utah, 2; Washington, 1; Oregon, 1.

<sup>3</sup> Period ended earlier than Saturday.

<sup>4</sup> Typhus fever, week ended May 20, 1939, 41 cases as follows: South Carolina, 3; Georgia, 14; Florida, 5; Alabama, 9; Louisiana, 1; Texas, 9.

<sup>5</sup> Colorado tick fever, week ended May 20, 1939, Colorado, 9 cases.

**SUMMARY OF MONTHLY REPORTS FROM STATES**

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week:

State	Men- gitis, menin- gococ- cus	Diph- theria	Influ- enza	Ma- ria	Meas- les	Pe- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid and paraty- phoid fever
<i>March 1939</i>										
South Carolina.....		144	4,831	423	102	122	5	22	1	30
<i>April 1939</i>										
Alabama.....	7	37	5,353	130	788	21	3	47	7	23
California.....	8	84	451	17	12,863	8	7	748	90	17
Illinois.....	11	133	246	2	128	2	5	2,038	57	16
Kentucky.....	6	32	398	6	88	2	2	247	18	14
Maryland.....	2	6	62	1	1,799	3	0	168	0	8
Michigan.....	8	40	88	1	1,654	-----	1	1,858	48	8
Minnesota.....	2	16	20	1	2,173	-----	0	311	35	7
Mississippi.....	1	21	11,538	1,857	3,355	407	2	9	2	6
New Jersey.....	4	46	30	2	163	-----	1	738	0	11
New Mexico.....	10	9	64	-----	117	-----	0	46	5	6
New York.....	16	56	-----	12	7,025	-----	8	2,348	1	34
North Dakota.....	3	4	227	-----	230	-----	0	46	27	4
Oklahoma.....	3	27	1,274	87	1,532	9	1	124	180	8
Rhode Island.....	1	1	-----	-----	172	-----	0	72	0	2
South Carolina.....		81	4,175	784	186	228	28	18	1	16
Tennessee.....	8	26	1,495	27	372	20	3	295	24	7

*March 1939*

Cases	
South Carolina:	
Chickenpox.....	213
Diarrhea.....	342
Dysentery (amoebic).....	1
German measles.....	25
Hookworm disease.....	112
Mumps.....	203
Ophthalmia neonatorum.....	3
Rabies in animals.....	27
Septic sore throat.....	8
Tetanus.....	1
Tularaemia.....	2
Typhus fever.....	6
Undulant fever.....	1
Whooping cough.....	378

*April 1939*

Anthrax:	
New Jersey.....	1
New York.....	2
Chickenpox:	
Alabama.....	200
California.....	3,727
Illinois.....	1,726
Kentucky.....	218
Maryland.....	319
Michigan.....	792
Minnesota.....	333
Mississippi.....	792
New Jersey.....	1,165
New Mexico.....	82
New York.....	2,639
North Dakota.....	44
Oklahoma.....	232
Rhode Island.....	140
South Carolina.....	218
Tennessee.....	205
Conjunctivitis, infectious:	
New Mexico.....	8
Dengue:	
South Carolina.....	5
Diarrhea:	
Maryland.....	8
South Carolina.....	581
Dysentery:	
California (amoebic).....	9
California (bacillary).....	19
Illinois (amoebic).....	3

*April 1939—Continued*

Dysentery—Continued.	Cases
Illinois (amoebic car- riers).....	15
Illinois (bacillary).....	11
Kentucky (amoebic).....	2
Kentucky (bacillary).....	1
Maryland (amoebic).....	1
Maryland (bacillary).....	9
Maryland (unspecified).....	4
Michigan (amoebic).....	1
Michigan (bacillary).....	1
Minnesota (amoebic).....	1
Mississippi (amoebic).....	226
Mississippi (bacillary).....	460
New Jersey (amoebic).....	1
New Mexico (amoebic).....	1
New York (amoebic).....	4
New York (bacillary).....	37
Oklahoma (amoebic).....	2
Oklahoma (bacillary).....	14
Tennessee (amoebic).....	2
Tennessee (bacillary).....	3
Encephalitis, epidemic or lethargic:	
Alabama.....	2
California.....	3
Illinois.....	7
Minnesota.....	2
New Mexico.....	3
New York.....	10
North Dakota.....	2
Oklahoma.....	1
South Carolina.....	3
Tennessee.....	2
Food poisoning:	
California.....	32
Maryland.....	1
German measles:	
Alabama.....	3
California.....	149
Illinois.....	38
Maryland.....	11
New Jersey.....	47
New Mexico.....	1
New York.....	133
North Dakota.....	18
Rhode Island.....	3
South Carolina.....	22
Tennessee.....	20

*April 1939—Continued*

Granuloma, coccidioidal:	Cases
California.....	7
Hookworm disease:	
Mississippi.....	469
Oklahoma.....	1
South Carolina.....	287
Impetigo contagiosa:	
Illinois.....	16
Maryland.....	10
Oklahoma.....	5
Tennessee.....	7
Jaundice, epidemic:	
California.....	7
Michigan.....	8
Leprosy:	
Illinois.....	1
Mumps:	
Alabama.....	221
California.....	3,541
Illinois.....	610
Kentucky.....	301
Maryland.....	332
Michigan.....	95
Mississippi.....	359
New Jersey.....	688
New Mexico.....	19
North Dakota.....	15
Oklahoma.....	27
Rhode Island.....	258
South Carolina.....	280
Tennessee.....	97
Ophthalmia neonatorum:	
Alabama.....	3
Illinois.....	3
Maryland.....	1
New Jersey.....	6
New Mexico.....	1
New York.....	13
South Carolina.....	3
Tennessee.....	4
Psittacosis:	
Oklahoma.....	1
Puerperal septicemia:	
Mississippi.....	25
New Mexico.....	4
Tennessee.....	2

## Summary of monthly reports from States—Continued

April 1939—Continued.		April 1939—Continued		April 1939—Continued.	
<b>Rabies in animals:</b>	<b>Cases</b>	<b>Tetanus—Continued.</b>	<b>Cases</b>	<b>Undulant fever—Con.</b>	<b>Cases</b>
Alabama	21	Michigan	1	Kentucky	8
California	127	New York	1	Maryland	6
Illinois	33	South Carolina	1	Michigan	11
Michigan	2	<b>Trachoma:</b>		Mississippi	2
Minnesota	1	California	22	New Jersey	8
Mississippi	15	Illinois	27	New Mexico	1
New Jersey	58	Minnesota	1	New York	21
New Mexico	23	Mississippi	2	Oklahoma	83
New York	6	New Mexico	2	South Carolina	1
South Carolina	18	North Dakota	4	<b>Vincent's infection:</b>	
<b>Rocky Mountain spotted fever:</b>		Oklahoma	10	Illinois	33
California	2	Tennessee	2	Maryland	5
Tennessee	1	<b>Trichinosis:</b>		Michigan	9
<b>Septic sore throat:</b>		California	2	New York	71
California	22	New Jersey	1	Oklahoma	10
Illinois	13	New York	1	Tennessee	94
Kentucky	22	<b>Tularaemia:</b>		<b>Whooping cough:</b>	
Maryland	21	Alabama	3	Alabama	180
Michigan	41	California	1	California	838
Minnesota	10	Illinois	3	Illinois	984
New Jersey	60	Kentucky	2	Kentucky	47
New Mexico	2	Mississippi	1	Maryland	94
New York	185	Oklahoma	7	Michigan	666
Oklahoma	77	South Carolina	1	Minnesota	165
Rhode Island	21	Tennessee	9	Mississippi	1,129
South Carolina	2	<b>Typhus fever:</b>		New Jersey	1,146
Tennessee	30	Alabama	13	New Mexico	79
<b>Tetanus:</b>		New York	4	New York	1,827
Alabama	7	South Carolina	9	North Dakota	30
California	5	Tennessee	2	Oklahoma	13
Illinois	1	<b>Undulant fever:</b>		Rhode Island	302
Maryland	3	California	21	South Carolina	436
		Illinois	19	Tennessee	141

<sup>1</sup> Exclusive of New York City.

## PLAGUE INFECTION IN CALIFORNIA AND WASHINGTON

### IN GROUND SQUIRRELS IN VENTURA COUNTY, CALIF.

Under date of May 13, 1939, Dr. W. M. Dickie, State Director of Public Health of California, reported plague infection proved in three ground squirrels, *C. beecheyi*, submitted to the laboratory on April 18, 1939, from a ranch on Casitas Pass Road, Ventura County, Calif., 2 miles east of the Santa Barbara County line.

### IN FLEAS FROM GROUND SQUIRRELS IN LINCOLN COUNTY, WASH.

Under date of May 13, 1939, Senior Surgeon C. R. Eskey reported plague infection proved in a pool of 44 fleas collected from four ground squirrels, *C. townsendi*, shot April 25 at a location 4 miles west of Rocky Ford and 17 miles northwest of Ritzville, Lincoln County, Wash.

**WEEKLY REPORTS FROM CITIES**

*City reports for week ended May 13, 1939*

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases		Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small-pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
	Cases	Deaths	Cases	Deaths								
<b>Data for 50 cities:</b>												
5-year average	158	107	46	6,988	663	2,231	19	413	27	1,407		
Current week	94	109	39	4,521	438	1,438	32	408	22	1,155		
<b>Maine:</b>												
Portland	0		0	0	2	2	0	1	0	20		24
<b>New Hampshire:</b>												
Concord	0		0	0	1	0	0	0	0	0		10
Nashua	0		0	0	1	0	0	0	0	0		6
<b>Vermont:</b>												
Barre	0		0	0	0	1	0	2	0	25		2
Burlington	0		0	1	0	0	0	0	0	2		9
Rutland	0		0	0	0	0	0	0	0	0		3
<b>Massachusetts:</b>												
Boston	0		0	160	18	56	0	8	0	14		219
Fall River	0		0	1	0	5	0	1	0	0		23
Springfield	0		0	35	1	2	0	1	0	1		38
Worcester	0		0	7	4	10	0	1	0	40		47
<b>Rhode Island:</b>												
Pawtucket	1		0	19	0	2	0	0	0	0		19
Providence	0		0	56	2	6	0	0	0	94		64
<b>Connecticut:</b>												
Bridgeport	0		0	19	2	6	0	1	0	0		36
Hartford	0		0	33	5	8	0	0	0	1		40
New Haven	0		0	396	1	6	0	0	0	15		26
<b>New York:</b>												
Buffalo	0		0	173	11	31	0	5	0	36		169
New York	23	12	2	265	83	259	0	97	3	98		1,551
Rochester	0	2	0	135	4	20	0	0	0	5		66
Syracuse	1		0	267	3	6	0	1	0	50		44
<b>New Jersey:</b>												
Camden	7		0	0	3	9	0	0	0	1		31
Newark	0	1	0	4	5	58	0	7	2	53		112
Trenton	0		0	0	1	21	0	3	0	3		26
<b>Pennsylvania:</b>												
Philadelphia	2	4	2	59	26	49	0	32	2	87		537
Pittsburgh	6	5	1	1	18	29	0	10	0	29		196
Reading	0		0	4	2	1	0	1	0	0		29
Scranton	0			0		15	0		0	0		
<b>Ohio:</b>												
Cincinnati	0	3	0	1	9	50	0	12	0	2		141
Cleveland	0	24	1	14	12	64	0	10	0	52		183
Columbus	0		0	2	4	12	3	4	0	2		106
Toledo	0		0	8	5	18	1	4	0	33		75
<b>Indiana:</b>												
Anderson	0		0	1	0	1	1	0	0	0		3
Fort Wayne	0		0	0	0	6	0	0	0	2		30
Indianapolis	0		2	4	6	43	2	5	0	40		105
Muncie	0		0	0	0	2	0	0	0	0		6
South Bend	0		0	2	1	0	0	0	0	10		10
Terre Haute	0		1	0	1	1	0	0	0	0		21
<b>Illinois:</b>												
Alton	0		0	0	1	3	0	0	0	2		7
Chicago	15	4	3	16	23	220	1	44	2	66		701
Elgin	0		0	0	0	5	0	0	0	1		4
Moline	0		0	1	0	0	0	0	0	2		12
Springfield	0		0	0	1	3	0	0	0	5		26
<b>Michigan:</b>												
Detroit	3		5	38	9	126	0	20	1	63		250
Flint	1		0	24	1	18	0	1	0	0		13
Grand Rapids	0		0	1	1	21	0	1	0	0		31
<b>Wisconsin:</b>												
Kenosha	0		0	1	1	4	0	0	0	3		15
Madison	1			103	0	4	0	0	0	7		16
Milwaukee	0	2	2	7	9	33	0	4	0	31		123
Racine	0		0	2	0	5	0	0	0	7		12
Superior	0		1	3	0	1	0	0	0	0		10

1 Figures for Boise estimated; report not received.

## City reports for week ended May 13, 1930—Continued

State and city	Diphtheria cases		Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever case.	Whooping cough cases	Deaths, all causes
	Cases	Deaths	Cases	Deaths								
<b>Minnesota:</b>												
Duluth	0	0	0	0	0	2	3	0	2	0	3	23
Minneapolis	1	0	98	8	8	8	8	4	2	0	22	114
St. Paul	0	3	3	71	10	12	0	5	1	1	5	75
<b>Iowa:</b>												
Cedar Rapids	0	0	1	0	0	0	0	0	0	0	6	0
Davenport	0	0	0	3	0	3	11	0	0	0	0	0
Des Moines	1	0	2	0	32	0	4	0	0	0	1	37
Sioux City	0	0	3	6	0	0	0	0	0	0	1	0
Waterloo	2	0	0	16	0	0	0	0	0	0	0	0
<b>Missouri:</b>												
Kansas City	0	0	3	8	12	5	15	5	0	0	1	95
St. Joseph	0	0	0	3	1	0	1	1	0	0	0	24
St. Louis	2	0	0	8	39	0	6	0	0	0	14	204
<b>North Dakota:</b>												
Fargo	0	0	1	0	0	0	0	0	0	0	0	9
Grand Forks	0	0	0	0	0	0	0	0	0	0	0	0
Minot	0	0	0	0	0	0	0	0	0	0	0	8
<b>South Dakota:</b>												
Aberdeen	0	0	202	0	0	5	0	0	0	0	0	0
<b>Nebraska:</b>												
Omaha	0	2	3	3	1	5	0	0	0	0	0	62
<b>Kansas:</b>												
Lawrence	0	0	0	0	0	0	0	0	0	0	0	4
Topeka	0	0	1	4	7	0	0	0	0	0	3	11
Wichita	0	0	8	1	5	0	0	0	0	0	1	26
<b>Delaware:</b>												
Wilmington	0	0	7	2	0	0	0	0	0	0	0	35
<b>Maryland:</b>												
Baltimore	1	0	154	16	16	0	12	0	0	13	257	
Cumberland	0	0	0	0	0	0	1	0	0	0	0	8
Frederick	0	0	0	0	0	0	0	0	0	0	0	3
<b>Dist. of Col.:</b>												
Washington	3	2	2	412	11	12	0	10	0	33	151	
<b>Virginia:</b>												
Lynchburg	0	0	94	2	4	0	0	0	0	11	15	
Norfolk	0	4	49	1	0	0	4	0	0	0	0	29
Richmond	0	1	318	3	2	0	0	0	0	0	0	40
Roanoke	0	0	0	0	2	0	1	0	0	0	0	17
<b>West Virginia:</b>												
Charleston	0	1	0	0	3	0	0	0	0	0	0	25
Huntington	1	0	1	0	0	0	0	0	0	0	0	0
Wheeling	0	0	0	1	1	0	0	0	0	10	29	
<b>North Carolina:</b>												
Gastonia	0	0	0	0	0	0	0	0	0	0	0	0
Raleigh	0	0	1	0	0	0	0	0	0	0	5	7
Wilmington	1	0	1	1	0	0	0	0	0	6	15	
Winston-Salem	0	0	9	1	0	0	2	0	0	0	0	9
<b>South Carolina:</b>												
Charleston	0	13	1	0	1	0	0	1	0	5	22	
Greenville	0	0	0	5	0	0	2	0	0	2	31	
<b>Georgia:</b>												
Atlanta	2	7	2	0	7	3	0	6	0	0	0	79
Brunswick	0	0	0	3	2	0	0	0	0	5	7	
Savannah	0	3	0	3	3	0	0	1	1	13	31	
<b>Florida:</b>												
Miami	0	2	1	0	4	1	0	4	0	2	33	
Tampa	0	0	0	55	2	2	0	0	0	3	27	
<b>Kentucky:</b>												
Ashland	0	3	0	0	1	0	0	0	0	0	0	7
Covington	0	0	0	1	2	2	0	2	0	0	0	10
Lexington	0	0	0	0	0	3	0	0	0	0	0	19
Louisville	0	0	0	10	1	6	0	6	0	8	69	
<b>Tennessee:</b>												
Knoxville	0	1	1	2	6	0	0	0	0	0	0	22
Memphis	0	1	0	1	2	14	0	4	2	12	73	
Nashville	0	0	1	1	17	0	4	0	5	49		
<b>Alabama:</b>												
Birmingham	0	3	0	2	4	0	0	4	0	5	57	
Mobile	0	2	0	1	1	0	0	0	0	2	17	
Montgomery	0	0	0	0	0	0	0	0	0	0	0	
<b>Arkansas:</b>												
Fort Smith	0	0	4	0	0	0	0	0	0	0	0	
Little Rock	0	0	0	4	1	0	1	0	0	17		

City reports for week ended May 13, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small-pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Louisiana:											
Lake Charles.....	0		0	9	2	0	0	0	0	0	7
New Orleans.....	5	2	1	27	10	5	0	6	6	3	140
Shreveport.....	0		0	10	2	0	1	2	0	1	37
Oklahoma:											
Oklahoma City.....	0		0	0	3	2	1	2	0	0	44
Tulsa.....	0			31		1	0		0	0	
Texas:											
Dallas.....	1			34	1	2	0	3	0	6	56
Fort Worth.....	0		0	26	2	2	0	1	0	0	47
Galveston.....	0		0	1	0	0	0	1	0	1	13
Houston.....	4		3	17	1	0	0	3	1	1	67
San Antonio.....	6		0	1	8	0	0	8	0	2	77
Montana:											
Billings.....	0		0	0	0	1	0	0	0	0	8
Great Falls.....	0		0	119	0	0	0	0	0	0	6
Helena.....	0		0	2	0	4	0	0	0	0	5
Missoula.....	0		0	13	0	0	0	0	0	0	3
Idaho:											
Boise.....											
Colorado:											
Colorado Springs.....	0		0	10	2	2	0	2	0	5	12
Denver.....	7		1	46	8	16	0	4	0	21	95
Pueblo.....	0		0	176	6	0	0	0	0	16	14
New Mexico:											
Albuquerque.....	0		0	0	1	2	0	3	0	0	11
Utah:											
Salt Lake City.....	0		0	11	3	5	0	0	0	9	29
Washington:											
Seattle.....	0		0	374	0	5	0	2	0	6	92
Spokane.....	0	1	1	172	3	2	0	0	0	0	30
Tacoma.....	0		0	3	1	1	1	1	0	0	33
Oregon:											
Portland.....	0	1	0	3	6	7	3	1	0	2	84
Salem.....	0	2		1		0	0		0	3	
California:											
Los Angeles.....	8	11	1	427	7	28	0	20	0	32	380
Sacramento.....	1	1	1	54	1	3	0	2	1	0	25
San Francisco.....	0	2	0	32	5	12	0	16	0	2	150

State and city	Meningitis, meningococcus		Polio-myelitis cases	State and city	Meningitis, meningococcus		Polio-myelitis cases				
	Cases	Deaths			Cases	Deaths					
Massachusetts:											
Boston.....	1	0	0	Maryland:							
New York:											
Buffalo.....	0	1	0	Baltimore.....	1	1	0				
New York.....	3	0	1	South Carolina:							
Pennsylvania:											
Pittsburgh.....	1	0	0	Charleston.....	0	0	16				
Ohio:											
Cleveland.....	1	0	0	Florida:							
Illinois:											
Chicago.....	1	1	0	Miami.....	1	1	1				
Tennessee:											
Texas:											
Knoxville.....											
Dallas.....											
Houston.....											

*Encephalitis, epidemic or lethargic.*—Cases: New York, 2; Philadelphia, 1; Columbus, 1; Memphis, 1.  
*Pellagra.*—Cases: Baltimore, 1; Lynchburg, 1; Wilmington, N. C., 3; Savannah, 1; Fort Smith, 1; New Orleans, 1.  
*Typhus fever.*—Cases: Savannah, 1; Mobile, 1; Houston, 2.

## FOREIGN AND INSULAR

### CANADA

*Provinces—Communicable diseases—Week ended April 29, 1939.*—  
During the week ended April 29, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis					5					5
Chickenpox		4		138	167	21	23	8	70	426
Diphtheria				42	4	6	5			57
Dysentery					2					2
Influenza		58			124		37			285
Lethargic encephalitis					2					2
Measles		12		498	746	3		13	2	1,274
Mumps		1		36	120	49			9	215
Pneumonia		5			20	1	2		12	40
Polio-myelitis								2		2
Scarlet fever			21	50	167	11	18	17	12	286
Smallpox					4					4
Trachoma						1				1
Tuberculosis		28	7	36	42	5			8	126
Typhoid and paratyphoid fever			1	8	2		2		1	14
Whooping cough		8	4	49	154	83	24	9	106	382

### ITALY

*Communicable diseases—4 weeks ended February 26, 1939.*—During the 4 weeks ended February 26, 1939, cases of certain communicable diseases were reported in Italy as follows:

Disease	Jan. 30-Feb. 5	Feb. 6-12	Feb. 13-19	Feb. 20-26
Anthrax	14	5	11	10
Cerebrospinal meningitis	37	43	37	53
Chickenpox	369	290	244	392
Diphtheria	630	593	569	518
Dysentery (amoebic)	10	5	13	10
Hookworm disease	26	10	10	23
Lethargic encephalitis	3	1	2	
Measles	1,391	1,356	1,371	1,467
Mumps	257	198	226	257
Paratyphoid fever	49	47	40	50
Pellagra	2			2
Polio-myelitis	30	24	13	26
Puerperal fever	84	36	33	34
Scarlet fever	229	223	221	174
Typhoid fever	277	259	249	287
Undulant fever	70	60	79	73
Whooping cough	321	352	307	358

## JAMAICA

*Communicable diseases—4 weeks ended May 13, 1939.*—During the 4 weeks ended May 13, 1939, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox.....	11	60	Puerperal sepsis.....	-----	4
Diphtheria.....	2	2	Tuberculosis.....	35	88
Dysentery.....	3	1	Typhoid fever.....	4	47
Erysipelas.....	-----	1			

## YUGOSLAVIA

*Communicable diseases—4 weeks ended April 23, 1939.*—During the 4 weeks ended April 23, 1939, certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax.....	23	2	Paratyphoid fever.....	11	-----
Cerebrospinal meningitis.....	136	30	Poliomyelitis.....	2	-----
Diphtheria and croup.....	471	44	Scarlet fever.....	233	4
Dysentery.....	11	-----	Sepsis.....	9	5
Erysipelas.....	141	1	Tetanus.....	20	10
Favus.....	4	-----	Typhoid fever.....	123	19
Lethargic encephalitis.....	1	1	Typhus fever.....	83	1

## CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for May 26, 1939, pages 711-723. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

## Cholera

*China.*—During the week ended May 13, 1939, 4 cases of cholera were reported in Hong Kong, China. Under date of May 20, 1939, Japan reported the presence of cholera in Canton, Fatshan, Whampoa, Honan, and Hainan Island, China.

## Plague

*Belgian Congo—Virakwa.*—During the week ended May 13, 1939, 4 cases of plague were reported in Virakwa, Belgian Congo.

*Ecuador—Chimborazo Province.*—According to information dated May 10, 1939, plague was reported in Chimborazo Province, Ecuador, as follows: March 1, 3 cases of bubonic plague at Alausi; March 15, 3 cases of bubonic plague at San Antonio; April 2, 8 cases of pneumonic plague at Pishillig; April 4, 7 cases of bubonic plague at Gonzal; April 10, 3 cases of pneumonic plague at Atapo. The report also states that a serious epidemic of pneumonic plague occurred on a

plantation near Columbe about April 25, 1939. Reference is also made to a previous outbreak of pneumonic plague which occurred in Riobamba during the period January 20 to February 14, 1939, in which 18 patients in a hospital contracted the disease, 17 of whom died.

*India—Calicut.*—During the week ended May 13, 1939, 1 case of plague was reported in Calicut, India.

*Siam—Bichitr Province.*—During the week ended May 13, 1939, 3 cases of plague were reported in Bichitr Province, Siam.

*United States.*—A report of plague-infected squirrels in Ventura County, California, and of plague-infected fleas in Lincoln County, Washington, appears on page 961 of this issue of PUBLIC HEALTH REPORTS.

#### Yellow Fever

*Brazil.*—Deaths from yellow fever have been reported in Brazil as follows: Espirito Santo State—Cachoeira, April 25, 1; Castelo, April 20, 1; Lambari, April 20–22, 2; Muniz Freire, April 14, 1, April 22, 1; Muqui, April 15–17, 2; Rio Pardo, April 14, 1; Sabino Pessoa, April 16, 1; Siqueira Campos, April 20, 1. Minas Geraes State—Manhãssu, April 20, 1.

*Ivory Coast—Guiglo.*—On May 17, 1939, 1 case of yellow fever was reported in Guiglo, Ivory Coast.